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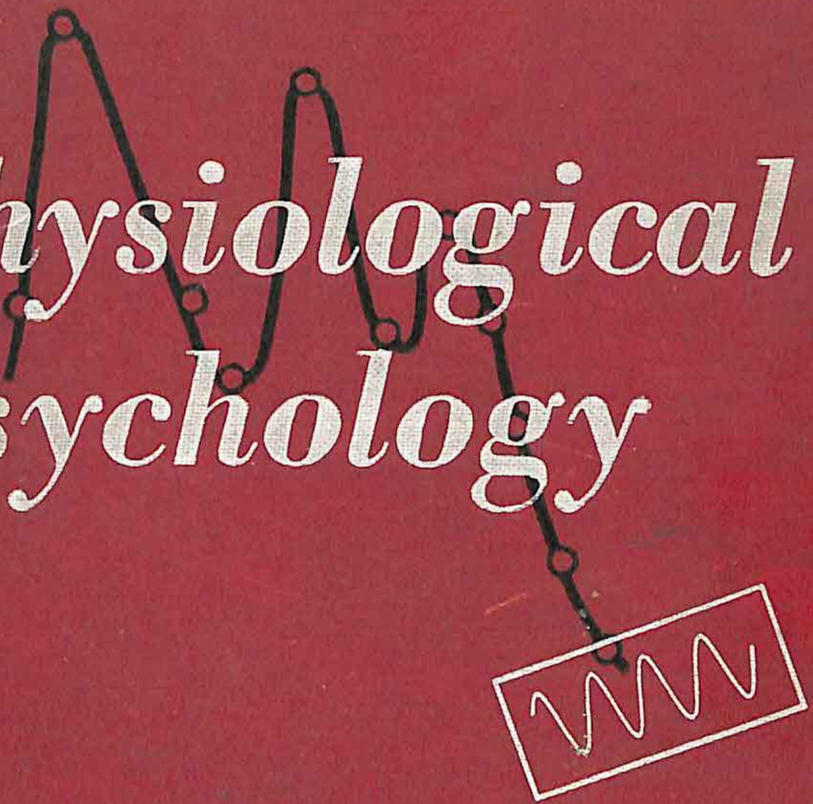

Physiological Psychology

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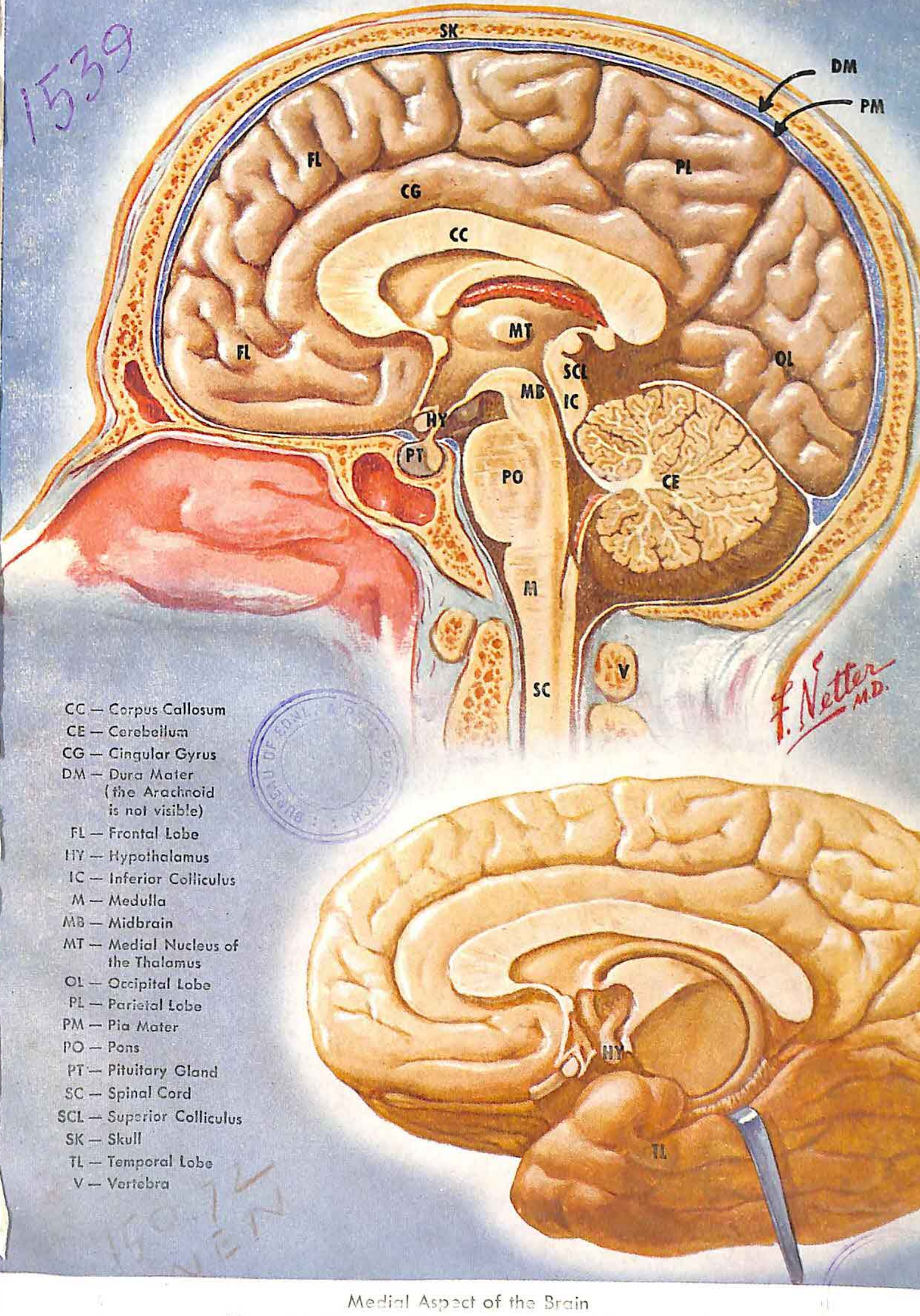

CC — Corpus Callosum
CE — Cerebellum
CG — Cingular Gyrus
DM — Dura Mater (the Arachnoid is not visible)
FL — Frontal Lobe
HY — Hypothalamus
IC — Inferior Colliculus
M — Medulla
MB — Midbrain
MT — Medial Nucleus of the Thalamus
OL — Occipital Lobe
PL — Parietal Lobe
PM — Pia Mater
PO — Pons
PT — Pituitary Gland
SC — Spinal Cord
SCL — Superior Colliculus
SK — Skull
TL — Temporal Lobe
V — Vertebra

Medial Aspect of the Brain
(Copyright *The Ciba Collection of Medical Illustrations.*)

Physiological Psychology

M. A. WENGER / F. N. JONES / M. H. JONES
University of California, Los Angeles

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Preface

This text was written with a specific course in mind — a second course in introductory psychology. As such, it presumes at least one semester of prior study in general psychology.

The point of view basic to the preparation of the text is that psychology is fundamentally a biological science. The student would seldom gain much of a physiological point of view, however, from the typical text used in an introductory course in psychology during the last decade or so. Although exceptions may be found, a general tendency in recent textbook writing for the beginning student in psychology has been to minimize treatment of the nervous system, action systems, and the specialized senses. Because of this comparative neglect it is often possible for advanced students to approach the study of, let us say, abnormal psychology, with little real knowledge or insight concerning many structures and functions that will be dealt with in the course. It is possible, furthermore, for a student to engage in a specialized study of such a topic as motivation without any real understanding of basic biological conditions of the organism and the drives which they induce. Moreover, there is a terminology which is used in many advanced psychology courses which the student can only derive from some consideration of bodily structures and related functions.

One solution to the problem posed by these conditions would be to establish a college course in physiology or biology as a prerequisite for the study of psychology, but few teachers of psychology would be satisfied with the emphasis upon structures and functions of organs common to most textbooks on physiology and would hold that the major emphasis should be upon functions of organisms — in other words, that the physiology or biology should be psychologically orientated. Furthermore, they would hold that only a part of the traditional physiology is germane to a study of psychology. At the present, therefore, it seems desirable to us to provide certain biological background information and terminology for the student as a part of his introduction to general psychology. It is our hope that the study of these subjects will better prepare the student for his later, more advanced, studies.

Since one of the stated purposes of this text is to enlarge the working vocabulary of the student, we have believed it expedient to include a glossary. As each significant term is introduced in context, whether or not it is explained at that time, it is shown in bold face type. Each word so shown is concisely defined in the glossary.

The authors' bias toward elementary textbook writing probably should be included in this prefatory statement. Although we feel that elementary texts should be largely factual in character and should forego the satisfactions to be derived from theoretical and controversial discussions, we occasionally find it impossible to confine ourselves to facts or to traditional ways of handling certain topics. The major portion of this text is confined to fundamental concepts on established facts in the biological sciences and, as such, has not seemed to us to require extensive documentation. When particular theories or when new or controversial material has been presented, it has been documented by references (see Appendix, p. 435). Occasionally the authors have taken the liberty of introducing new interpretations, and in one chapter, that dealing with emotional behavior, the treatment is new and much of it is theoretical.

Although all authors have participated in the revision and checking of all the chapters, it is true that where a theoretical position or bias was strong, the remaining authors tended to defer to the wishes of the author responsible for the original draft of a given chapter. In general, M. A. W. was responsible for material related to motivation, emotion, and the internal patterns of activity; F. N. J. for the central nervous system and total bodily integration; and M. H. J. for sensation.

We wish to thank the instructors who have used this text in its mimeographed form over the past few years and who have helped improve it in many ways. These instructors are Drs. John Helmick, Irving Maltzman, John Michael, and John Schwarz. Professor H. F. Harlow has read and painstakingly criticized the entire manuscript except for Chapter 25 and the glossary. Many improvements are due to his suggestions, but any inaccuracies or unusual theoretical interpretations remaining in the text are not to be ascribed to him. Our thanks are due the authors and publishers who have kindly permitted us to use illustrations. Appropriate acknowledgement is given with each figure. Finally, we must acknowledge our debt to the several thousand students who have been instructed from this text in one mimeographed form or another. Their comments have been very helpful.

Los Angeles, Calif.
March, 1956

M. A. W.
F. N. J.
M. H. J.

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1 *Introduction*

In these days of rapidly increasing interest in psychology, many students dabble in the subject. For most, their study is casual and largely motivated by their interest in themselves and in their fellow men. Although a sampling of the world's knowledge is not to be discouraged, this volume is not intended for the sampler. Instead, it is designed for those of you who, having sampled psychology, have decided on more thorough study. As such, it has two major purposes: (1) to acquaint you with some of the fundamentals of a study of human behavior which are usually eliminated, or only briefly treated, in introductory courses; and (2) to provide a more adequate vocabulary for your further study of human behavior.

The Importance of Vocabulary. The importance of acquiring a technical vocabulary for your further study of psychology cannot be overemphasized. In the fields of human activity, confusions which arise from different interpretations of words produce new problems which lead to more words, in turn subject to misinterpretation. Some writers believe that many of the world's ills are basically semantic; that is, they arise out of misinterpretation or misuse of words. Still others argue that many problems in personal adjustment may arise from the same source. It is certain that most of your increasing knowledge of human behavior will be derived from words; and that knowledge will be used by you and expressed to others in the form of words. You must learn those words well if you would be an effective student.

In order to assist you in your mastery of a working vocabulary in psychology, each new term with a technical meaning will appear in **bold face type** when it is introduced in this text. It will be shown again and concisely defined in the glossary at the end of the book.

Psychology is usually defined today as the science of the **behavior of organisms.** In other words, it involves a study of what organisms do, whether it be thinking, remembering, or moving. It might be argued, then, that these activities should be describable in terms of verbs and adverbs, but, as one introductory text puts it,

". . . like other sciences, psychology finds it convenient to transform its verbs into nouns. Then what happens? We forget that our nouns are merely substitutes for verbs and start hunting for the things denoted by the nouns — for substances, forces, faculties — but no such things exist; there is only the individual engaged in these different activities." (p. 19 (1))

Here, then, is another semantic problem. As you pursue your study of psychology, remember that its major subject matter lies in verbs and adverbs and suspect all nouns that do not denote parts of organisms, acts of organisms, or objects and forces of the environment. You will note an attempt to follow that point of view in this volume and you may be surprised at some of the new conceptions that result and at the sudden meaninglessness of some of the older concepts.

Specialization in Science. A living animal, especially a complex one such as man, displays a tremendous variety of activities. It eats, digests, gets about from one place to another, makes and uses tools, maintains its bodily temperature relatively constant, and does a very large number of things of which these are but a few examples. To study all of these activities at once is quite impossible. It is necessary that anyone interested in studying the activities of an organism decide just what aspects of the total he wishes to observe in a given situation, and devise techniques which permit the observation of those aspects with the greatest possible accuracy and precision. Since it is impossible for any one person to command all of the techniques necessary to study all aspects of activity, individual scientists have come to concern themselves with more or less limited kinds of activity, and a number of specialized sciences have arisen. Among many of the sciences, however, there is no natural division, and so interscience specialization also occurs. For example, both physical chemistry and physiological chemistry are well known to you, and you may have heard of such combinations as neurochemistry or geophysics. All of these represent areas of investigation which are at a borderline between two or more related sciences; and physiological psychology or psychophysiology is one of them.

Physiology and Psychology. With the development of specialization among the sciences, there has grown up a rough division of the study of the activities of the living animal into several disciplines, among which are **physiology** and psychology. It should be emphasized at once that this is not a natural division, but rather, as has been indicated above, it is a division of convenience. In general, those investigators who are identified as physiologists have chosen to study activities localized within the organism, and to deal with internal systems and changes; thus, physiology is sometimes defined as the study of **organ** and **tissue** functions. Typical physiological investigations are concerned with such problems as the processes of digestion, the chemistry and circulation of blood, and the characteristics of muscular contraction. Psychologists, on the other hand, are more concerned with activities which involve the total organism. Typical investigations of a psychological nature are studies of learning, of the development of personality, or of the effect of motivation. There is, however, no hard and fast line between these two fields. Psychologists have sometimes studied part activities, such as the neuromuscular activity in a single finger, the functions of parts of the eye, or the activity of a few segments of the spinal cord; and physiologists have studied such total organismic activities as homing in birds and orientation in the bat.

It is well to keep in mind that the general division of labor between the physiologist and the psychologist is partly a methodological one. We may expect the physiologist to be competent in techniques for the electrical recording of activity in a nerve, the biochemical assay of the by-products of nervous activity, or the changes induced in starches by the action of digestive enzymes. The psychologist, on the other hand, has at his command techniques for measuring learning, for assaying ability or efficiency, or for studying other organismic behavior familiar to the student who has had a beginning course in psychology. But the physiologist sometimes finds it necessary to pay attention to psychological controls when dealing, for example, with the effects of low oxygen intake on the human subject, or with effects of various drugs; and the psychologist must often be concerned with sensory end organs, or the physiological changes occurring in such behavior as drug addiction.

As inevitable outcomes of the different techniques used by physiologists and psychologists, there have grown up different sets of data and different sets of hypotheses to account for them. It may be said that if one asks a physiological question, that is, applies a physiological technique, he will receive a physiological answer. Conversely, if he asks a psychological question, he receives a psychological answer. Although dealing with the same organism, the two disciplines have different outcomes. However, the physiologist and the psychologist realize that they are abstracting limited aspects of the total activity of the organism, and that a complete picture must include the contributions of both groups.

Structure and Function. It may help the student to understand more clearly the relationship between physiology and psychology, and their underlying unity, if he realizes that these disciplines share the same animals for their investigation. Many times psychologists have studied rats in mazes or discrimination boxes, and then passed the animals on to physiologists who have used them in physiological experiments. Both groups of experimenters have used them in different ways and have usually studied different functions.

Although the primary interests of both physiologist and psychologist are functional, for both study activities, it is necessary for both to understand the anatomy of the organisms they study. In the first place, a knowledge of structural relationships makes the comprehension of functional relationships easier; and very often a supposed function may be questioned when no structural relationship can be demonstrated. If one does not know the nervous connections by which the brain can influence the lining of the stomach, he is likely to consider as rather questionable the idea that psychological conflicts can lead to ulcers. But the connections are there. For extrasensory perception (telepathy) , however, there are no demonstrated structures, and so most psychologists are skeptical of its existence.

In the second place, structure is studied because it may lead to useful predictions of function. At one time, neurology was largely the study of structure, and functional relationships were inferred from details of structure. Although this procedure cannot be used to the exclusion of physiological experimentation, many useful inferences have been, and still are being, drawn. The reverse is also true, and functional relationships give hints to structural relationships. Thus, a careful anatomical study of the nervous connections between eye and brain will lead to the conclusion that certain areas at the posterior end of the brain are concerned with vision. On the other hand, the discovery that when a light is flashed into the eye electrical activity may be detected in the posterior region of the brain leads to the conclusion that there is some nervous connection between the eyes and that region.

The third reason for interest in structure is that functional changes in the organism may lead to structural changes. For example, a diet deficient in vitamin D can lead to upset functional relationships within the body of a child, and result in various deformities of structure, the most prominent of which will be bowed legs. Or, a continued conflict situation may lead, by changing certain functional relationships, to structural changes in the stomach, in this case ulceration. Another interdiscipline known as **psychosomatic medicine** has grown out of the recognition of such relationships, and we shall discuss its relationship to psychology in a later chapter.

Physiological Psychology Defined. We may briefly define physiological psychology as *the study of relationships between bodily processes and behavior;* and, of course, it assumes the possibility of showing some type of correlation between a given psychological activity and some set of physiological activities within the organism studied. We find, for example, that visual perception in man is always accompanied by electrical activity in a certain portion of the brain, and that removal of this area through operation or injury will eliminate the possibility of visual perception. In this one example we can see psychological data (the reports of visual perception), physiological data (electrical activity in the brain), and anatomical data (structure of the brain) related in a single organic event. No single approach completely describes what is going on, and the physiological psychologist is interested in all of them.

The physiological psychologist in a very real sense, then, is concerned with the whole organism. His interest lies in the reintegration of the total activity of the organism and to accomplish this goal he must cross the boundary into physiology. In physiological psychology the student will find an opportunity to study the living organism as a functional unit, rather than as subdivided into physiology and psychology.

The Contributions of Physiological Psychology. The study of physiological psychology may be expected to contribute to the understanding of behavior in several ways. In the first place, it leads to the realization that an organism which is **perceiving** or **learning** is also engaged in very complex internal activity, much of which is related to the perceiving or learning. It gives a more complete picture of the living organism than does either physiology or psychology alone.

A second contribution made by physiological psychology is an extension of the first. Whether interested in the laboratory or the clinic, the student must come to realize that the organism is dynamic; that is, its internal processes are in constant change and contribute to the determination of an ever-changing stream of behavior. The organism is not a closed box provided with input and output terminals which one may study only by stimulating the input terminals and observing the output. Much of what goes on inside the organism is known and will help us understand the output and its true relations to input. Consider for a moment some examples from **psychopathology**, where toxic and other changes lead to behavioral disorders. Undulant fever, low blood sugar level, or syphilis of the brain may bring about behavioral changes which have been treated by psychotherapy because of insufficient information about the physiological changes occurring in the patient.

In the third place, the psychologist may derive hints from physiology which lead him to propose psychological processes not previously known, or to select one or another hypothesis concerning behavior on the basis of physiological evidence. For example, one group of psychologists, after assaying the deficits in the behavior of monkeys after the removal of certain brain tissue, remarked that there was no psychological category by means of which to describe the loss. This leads to the suggestion that there are psychological processes which we have not yet isolated. Again, it is possible to reject certain theories of pain and of hearing on the basis of physiological evidence, as we shall see later. Also, the psychologist has no faith in the doctrine of "faculties" or packaged units of generosity, truthfulness, talkativeness, or friendliness, partly because physiological studies of brain activity fail to demonstrate such isolated functions. In the last analysis, one may expect psychological and physiological concepts to be compatible.

The Development of Physiological Psychology. Physiological psychology developed out of physiology over 100 years ago. The interest of certain physiologists in the sense-organs led to experimentation on their functions and gradually it became obvious that such a study also involved problems not regarded as physiological in nature, such as the set or expectancy of the subjects, or previous practice. Other physiologists became interested in the functions of the brain and their experimentation required some attempt at a description of behavior. Over the years this need to describe and classify behavior and behavior defects due to brain damage has come to demand more precise techniques. A field of study gradually developed which was no longer pure physiology and which led to experimental psychology and some of its specializations.

As early as 1874 a textbook was published by a German physiologist named Wundt which was actually entitled *Principles of Physiological Psychology*, and which brought together what was known at the time regarding sensation, response, brain function, and many other topics which we still study. It is impossible to draw the line which separates historically the preliminary physiological work from the psychological, just as we find no clear-cut distinction between the two parent sciences today. Much experimental work by such men as Weber, Fechner, and Helmholtz preceded the publication of Wundt's text and still fills a significant place in modern psychology. Other kinds of research in physiology have also been important, such as studies of the cell initiated by Schwann, studies of reflex action by Sherrington and his followers, and the development of the doctrine of evolution following Darwin. All of these contributed knowledge and furnished powerful tools which we shall find indispensable in our attempt to understand the behavior of organisms.

Plan of the Text. In the first paragraph of this chapter it was stated that one of the major purposes of this text is to acquaint you with certain of the fundamentals of a study of human behavior. We are interested, of course, in what organisms do — so we must study muscle action. But to understand organismic action we must know the manner in which it is initiated and altered; therefore, we must study the functions of the sense organs and must discover all we can about the many conditions that influence activity. This, of course, involves a study of the functions of the nervous system, of many glands of the body, and of many chemical and physical conditions that alter the activities of nerve and muscle.

We have attempted to organize the text so that each chapter develops logically from those preceding and draws upon the material already learned. In so far as is possible, the book is organized as a single unit and any material which is skipped is likely to make later study more difficult to comprehend.

It should be pointed out, perhaps, that on many topics opinions differ, as is to be expected in any active field of scientific endeavor. The student should be prepared to find that other courses in psychology present different opinions and, also, that as new knowledge accumulates conclusions must be changed. But the basic point of view of this text is that a consideration of the physiological events related to behavior will better enable us to understand behavior itself. We, therefore, consider physiological psychology an important aspect of all fields of psychology.

Suggested Readings

1. Morgan, Clifford T. and Stellar, Eliot, *Physiological Psychology*. New York: McGraw-Hill, 1950, Chap. I.
2. Loucks, R. B., "The Contribution of Physiological Psychology," *Psychological Review,* Vol. 6, 1941, pp 105–126.

2 | *The Organism*

Darwin's theory of evolution and Schwann's cell theory gave tremendous impetus to the development of the biological sciences. Both of these theories suggested that insights into the complex nature of man could be gained by studying other animals, even those which consist of a single cell. Although the results of such studies always must be verified by direct observations on man, they contribute much to our understanding of human structure and function.

In the years before the dissection of human cadavers became a general practice, most of man's knowledge about his own anatomy was inferred from dissections of other animals; and the field of comparative anatomy is still an important contributor to scientific knowledge. Of greater significance for the lives of each of us is the field of comparative physiology because so much of what is known in medicine, or applied physiology, has been learned from investigations carried out with infrahuman animals. Psychologists also make frequent use of simpler animals in order to unravel some of the complexities of human behavior, and the field of comparative psychology is an important one which should not be neglected by the student who would understand his own behavior and that of his fellow men.

This text will make frequent use of the comparative approach, not only because physiology itself is so largely comparative in nature, but because it is often more effective to illustrate behavior by using animals simpler than man. In fact, by considering the activities of a single cell, such as amoeba, we may gain a relatively simple view of some of the fundamental characteristics of all animals, including man. This is possible because man and most other forms of animal life are multicellular organisms whose various parts together demonstrate all of the characteristics of single-celled organisms.

The differentiation of cells for special activities or functions is one of the most fascinating subjects in the study of evolution. There is some specialization of function even in amoeba, and other single-celled organisms show even more, such as the development of the hair-like cilia in paramecium by means of which it moves about in its environment. Such specialization is within the single cell, however, and only in multicellular organisms does one find true cellular differentiation. Here certain cells become highly specialized for certain kinds of functions, such as **nerve cells** or **muscle cells,** although they still retain many of the characteristics of the separate single-celled organism. When these differentiated cells are grouped in a similar fashion and serve a similar function, they are known as **tissue;** and when different tissues combine in a given structure, they are known as an **organ.** Physiology often is defined as the study of the functions of organs and organ systems (Chap. 1); but physiologists do not confine their attentions to such groups of cells and much of what they know about organic functions has been learned initially through studies of single cells **(cytology).** Psychologists also must occasionally direct their attention to single cells and, in this text, we shall have occasion to learn something about nerve cells of different sorts and about muscle and gland cells. First, however, let us consider the functions of the relatively simple and undifferentiated cell — amoeba; and learn something of the properties of the material of which it is composed — **protoplasm.**

Characteristics of Protoplasm. Since protoplasm is the matter of living cells, let us think in general terms and consider amoeba as a representative or relatively undifferentiated protoplasm. Even here, however, considerable differentiation occurs. Among the differentiated subcellular structures the one of most interest to students of psychology is the cell membrane which encloses the rest of the cellular protoplasm (i.e., the **cytoplasm**), since it is somewhat specialized for the three important functions that have most to do with adaptive behavior — **irritability, contractility,** and **conductivity.**

You know from your own experiences that man is sensitive or irritable to certain kinds of physical energy changes known as **stimuli.** For animals other than man, we infer irritability when the animal consistently does something, or reacts, following a given kind of stimulation. These reactions need not be openly observable, or **overt,** but may be occurring within the organism, i.e., be **covert,** and be detectable only by specialized instruments. Let us first consider the overt reactions of amoeba.

If this tiny organism be observed under a microscope and various sorts of stimuli applied, we shall see that two general kinds of **reaction** occur. One is away from the stimulus object and may be called an avoidance response. The other is toward the stimulus object and may be called an approach response. Since avoidance responses occur almost invariably to localized pressures, to electrical currents, to marked changes in temperature, and to acids, we say that protoplasm is irritable to certain forms of mechanical, electrical, thermal, and chemical stimuli. Since approach responses toward food and other particles occur only at times, we do not conclude that protoplasm is generally irritable to such particles. Instead we say that under certain circumstances such particles become effective stimuli for approach reactions — in this case, food taking. Such observations have been conducted under highly controlled conditions where it can be ascertained that no change in the external environment is responsible for the occurrence or lack of occurrence of approach reactions. We conclude, then, that some change in the protoplasm itself must account for the variability of these approach responses. We reason that an internal change must afford the condition under which a particle of matter acquires stimulus value, or that another kind of change sets the condition under which the same particle is ineffective in eliciting reaction. From such a conclusion, it is but a short step to the inference that changes within the organism must also account for the absence of avoidance behavior in the presence of stimuli which ordinarily elicit it. We shall have much to say later about variability of behavior in man and other animals, but here it is seen in probably its simplest form.

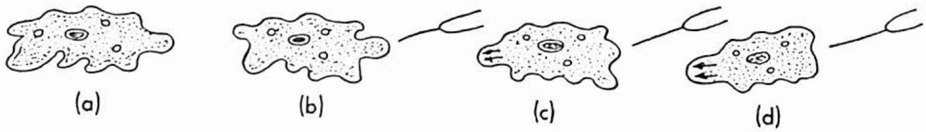


FIG. 2.1. Movements of amoeba away from stimuli. A pseudopod is produced on side opposite point of stimulation by a needle.

In addition to viewing the cell membrane of amoeba as the prototype of specialized sensory or **receptor cells** in man, we also may consider it as the prototype of specialized muscle and nerve cells. Figure 2.1 shows what happens when amoeba is stimulated with a needle. We see that the avoidance reaction occurs in a series of coordinated phases, which involve a relaxation of the membrane within a limited area opposite the site of stimulation and increased tension or contraction of the membrane in the area of stimulation. The result is an extension of a portion of amoeba, known as a pseudopod or false-foot, and a pulling of the remainder of the cytoplasm away from the stimulus. Motility in amoeba is accomplished by alternate contraction and relaxation of the cell membrane and related structures, whereas in man and other more complex animals it is accomplished by muscle cells which have become specialized for contraction.

The first marked change in the cell membrane (Fig. 2.1) is on the side opposite from that stimulated, which leads us to the third important property of protoplasm — conductivity. It is now known that the stimulus sets up an electrochemical change in the membrane itself which is propagated throughout the membrane in all directions from its source, much as the **nerve impulse** is propagated along a nerve cell. Whether or not this change is qualitatively the same as the nerve impulse in man, the cell membrane may be thought of as a conductor and as the prototype of nerve cells which have become specialized for conduction and thereby for the integration and coordination of organismic activity.

Other properties of protoplasm seem to involve the whole cell. Although future investigations in cellular physiology may result in attributing some of them to particular portions of the cytoplasm, we shall not discuss speculations concerning these possible relationships. It is important for us to know, however, that some of these properties are demonstrated by every living cell in our bodies and that some of them have much to do with the manner in which we behave. Four of these properties are: **metabolism, secretion, elimination,** and **growth.**

Metabolism may most simply be described as the process of life itself. It involves the taking of food, oxygen, and other materials; the digestion of food with the aid of various substances which are elaborated within the cell (which serves to illustrate the property of secretion) ; the assimilation of digested foodstuffs and their utilization for energy expenditure through the process of oxidation; the elimination of undigested matter and the excretion of other waste products of metabolism; and the recovery or repair of the cell after work or injury. When the cell is quite active and a relatively large amount of energy is being expended, a state of **catabolism** is said to predominate; whereas, during periods of rest and recovery, a state of **anabolism** predominates. These two processes in metabolism should not be thought of as mutually exclusive for both are continuous and coexistent in the living organism; so the term " predominates " should be remembered when they are used.

The property of growth, although shown by all protoplasm, is not well understood. In man and other vertebrates (see Chap. 15) it is related to certain chemical secretions, and its termination probably is related to the development of other secretions.

Another important characteristic of protoplasm is **reproductivity** and it, along with certain aspects of metabolism, best serves to differentiate living and nonliving matter. We may produce machines that demonstrate irritability, conductivity, and motility in such a manner that they are somewhat adaptable to their environments.

They may be made to produce certain agents that are needed for their operation and to eliminate waste products of their work; but they do not repair and restore their parts after damage and, more significant perhaps, they do not reproduce their own kind. In other words, we can artificially create most of the properties of protoplasm but we cannot (at the present, at least) create a living cell.

Reproductivity is unique among protoplasmic characteristics for still other reasons. For one thing, in most multicellular organisms it involves not only cellular differentiation but two remarkable forms of specialized cells — the male and female germ cells. Furthermore, the reproductive function of protoplasm is the only one that can be altered without modifying the **functional integrity** of an organism and, if the property of growth be excluded also, it may be said that each of the remaining properties is necessary for life itself. Let us turn our attention, then, to some of the conditions which make life possible — to some of the needs of protoplasm.

Needs of Protoplasm. When we consider the needs of protoplasm, or the needs of man, it is important to remember that "need" is a concept that always implies some goal, purpose, or end. This does not mean that we must ascribe " purpose " to amoeba. It means only that, if amoeba is to continue as a living organism, certain conditions are necessary. These conditions we speak of as " needs "; and since metabolism is the life-process itself, most of these needs are seen to be related to metabolism. Some of the major physiological needs of all animal protoplasm are listed in the following tabulation:

Some Major Needs of Animal Protoplasm

Foods – largely carbohydrates, fats, and proteins
Catalysts and other metabolic agents – oxygen, water, certain mineral salts, and enzymes
A certain range of temperatures
Periods of rest from activity – for restoration of functions
Elimination – of heat and other deleterious products of metabolism and ingestion
Protection from injury – from certain chemical, mechanical or electrical conditions, or from extreme temperatures

Organismic Function. Later, in a more detailed study of **motivation** (see Chap. 20), we shall see that these physiological needs are the basis for much of animal behavior, including that of man. For the present, let us consider only briefly what amoeba does about some of its needs, for one of the fundamental principles of biology is that living organisms act to satisfy their needs. If they do not do so successfully they die, hence do not reproduce their kind and, therefore, are known to us only as extinct forms.

Some of these acts or processes of need-satisfaction take place solely at a physical level and are thought of mainly as physical properties of cells. They involve an exchange of matter between the external environment and the cell through its membrane and are controlled largely by physical conditions on both sides of the membrane. For example, as oxidation occurs in the cell, two of its by-products, heat and carbon dioxide (CO_2), will accumulate and momentarily make the cell out of balance with its environment. Since the cell is a physical object, however, it will lose heat and CO_2 to the surrounding environment until temperature and CO_2 pressure are approximately equalized on both sides of the membrane and a state of balance or equilibrium is attained.

In these equilibratory exchanges between cell and environment the cell membrane plays the controlling role. It is selective of what may pass through it (and hence is spoken of as "semipermeable") and this selectivity varies with the internal condition of the cell and the nature of the environment.

Most of the substances which are found in solution within living cells are known as **electrolytes** because they tend to form **ions** carrying positive or negative electrical charges. Differences in ionic concentration may occur on the two sides of the cell membrane. This is the basis for the properties which we have called irritability and conductivity (see Chap. 3) and leads to the property called contractility.

Let us turn now to other activities of the cell and suppose that as CO_2 has accumulated within the cell we have arranged to increase it in the external environment. Now the cell could not lose this product to the environment, or it might even acquire more from the environment if our artificial increase has been greater than that occurring within the organism as a result of its own catabolic activities. What would happen? The answer is probably one of the most interesting that students of biology have ever found, for it serves to explain so much of the behavior of organisms. The cell would demonstrate increased motility which would continue until another more equable environment had been discovered or until the functions involved in motility began to fail and, given no adequate change in environment, death followed. This increased motility or reactivity of the organism is preceded, of course, by conductivity and by irritability to the stimulus situation of increased carbon dioxide. It will occur whenever the functional integrity of the organism depends upon avoiding some stimulus or stimulus condition if the organism is irritable to that condition. It may be seen in newborn human infants or in any organism which has not learned specific ways and means of satisfying its needs, of adjusting to its environment — i.e., of re-establishing a state of equilibrium.

Consider now the qualifying phrase in the above paragraph, "If the organism is irritable to that condition." This implies that organisms are not irritable to all conditions that are harmful to them, and this we know to be true. A study of the sensory equipment in man shows that there are many harmful conditions that we cannot know directly because we possess no receptor cells that are irritable to those conditions. For example, we are not directly aware of a need for oxygen, but are led to search for more adequate air only when CO_2 tension increases in our body cells and, therefore, in our blood. Cells that are sensitive to increased CO_2 tension take care of us rather well in our ordinary environments, but they do not help the high-altitude aviator, who has to be told when to put on his oxygen mask. It is obvious, however, that animals which live to reproduce their kind are irritable to most of the external or internal stimuli that would destroy their lives or interfere with their part-functions.

Such harmful stimuli are sometimes called **noxious.** We experience them as uncomfortable or painful, and sometimes we even just call them "irritating." In man, as well as in amoeba, they usually lead to avoidance reactions; and we say that man and amoeba need to eliminate or reduce these stimuli. Remember, however, that the word "need" is only conceptual — only a way of thinking about certain conditions — and that it is the stimulus conditions themselves that elicit behavior, given the proper organismic conditions.

When these stimulus conditions or unlearned irritating states are internal we refer to them as **need states,** or usually simply as **drives,** and we are less likely to regard the resulting behavior as avoidance behavior than when the stimulus is external. We already have mentioned approach reactions in connection with the food-taking response of amoeba; it should be pointed out also that some of our approach responses operate to remove the stimulus condition. Consider, for example, the approach of a male dog to a tree and the act of micturition which usually follows. Although the initial behavior is an appoach response, the total behavior pattern certainly has the effect of avoidance; it results in reduction or removal of the stimulus condition — of the drive (in this case, sensations from the bladder). We may think of a need for, or a drive toward, elimination; but the most significant observation is that the resultant behavior removes the internal stimulus condition.

We have already mentioned the need-satisfaction character of behavior, but we often prefer to think in terms of drive-reduction or stimulus-reduction. An even more useful concept in some instances is **equilibratory behavior,** and some psychologists prefer the term "adjustive behavior." The latter terms are quite broad, of course, and have the advantage of encompassing not only the purely physical exchanges that occur at the cell membrane, but also behavior of the variety we demonstrate in trying to win a football game. Regardless of the specific terminology employed in a particular discussion, it is possible to interpret all organismic behavior and internal part-function as though it were directed toward obtaining and maintaining a state of equilibrium, physically, physiologically, and psychologically. We then may consider external stimuli and internal drives, along with other physical changes affecting single-celled or multicelled organisms, and speak of all as disequilibrating factors or conditions.

It is important to remember, however, that physical equilibrium is not identical with physiological equilibrium and that neither is the exact counterpart of psychological equilibrium. We shall reserve until later any further discussion of psychological equilibrium, but a major distinction between physical and physiological equilibrium should be pointed out now. A set of scales in balance is in physical equilibrium and demonstrates no motion or activity except at a molecular level. A living organism may be in physical equilibrium with its environment and may be overtly immobile, but the process of metabolism continues and we shall see later that in complex organisms the activity of many parts is involved. We must think, then, of physiological equilibrium as involving a kind of balance among these functions. Two ways of referring to different aspects of this state should now be considered. One is known as functional integrity; the other is called **homeostasis.**

Maintenance of Functional Integrity. When we examine a living animal we observe that one manner in which it differs from a nonliving object is in its ability to maintain a reasonably constant functional relationship with its surroundings. Indeed, to survive at all, a living animal must stay in relative equilibrium with its environment. When the difficulties of the new environment become too great, this equilibrium is upset, and death results. A freshwater fish, for example, may adjust to moderate changes in the saltiness of the water in which it lives. Beyond a certain point, which depends on the given species of fish, equilibrium cannot be maintained; the fish loses water from its body cells to the environment and dies. Under one set of conditions, the fish was obviously alive, i.e., could maintain its functional integrity; under the other set of conditions it was obviously dead, i.e., irreversible chemical changes had taken place which did not represent the maintenance of equilibrium and which interfered with the integrated functions of its organs.

It will be seen at once that being alive is an active process. It is a term which we apply to bodies which display the property of maintaining functional integrity and does not refer to a thing or entity, " life," possessed by the organism. This is why we prefer to use the active form, living, rather than the noun, life. The properties of the living organism are functional properties. This will make it clearer why, in later chapters, the term " emotional behavior " will be preferred to " emotion," and " perceiving " to " perception."

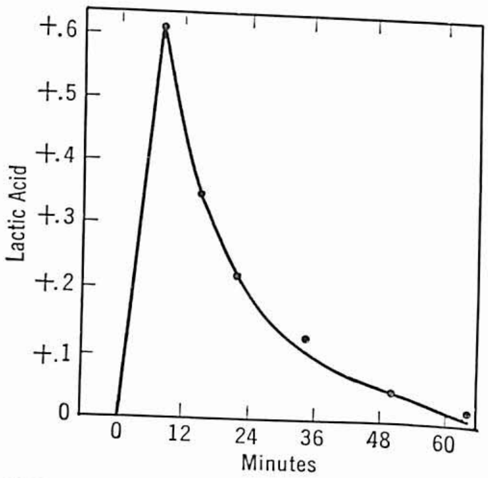


FIG. 2.2. Lactic acid in blood of man after injection during the first twelve minutes. It is almost completely cleared in one hour. (After Adolph.[1])

Homeostasis. The physiological aspect of this maintenance of equilibrium has come to be known as homeostasis. This term does not imply that the organism possesses a unique homeostatic property, but is descriptive of the general state of affairs. Some years ago the great physiologist Bernard recognized that constancy of the internal environment is necessary for free and independent life. In other words, the ability of the organism to maintain its internal processes relatively constant determines the range of environments in which it can survive. Here, for example, lies one of the secrets of the greater efficiency of mammals compared to reptiles. The mammal is better able to maintain its internal processes constant. It is a more efficient homeostatic mechanism because it can maintain internal temperature so much more nearly constant.

Unfortunately it is very easy to slip into the error of using homeostasis as an explanatory term. Does the blood tend to have a constant specific gravity? Homeostasis is responsible! Is a man's internal temperature relatively constant? Homeostasis maintains it! It is better to think of homeostasis as a concept derived from the facts of relative constancy and as being a convenient descriptive term for use in referring to a very large number of such facts, rather than as an explanatory principle.

A further point is that specific mechanisms, or patterns of mechanisms, will be found to underlie different homeostatic functions. An example of a homeostatic function which depends upon a biochemical basis is the clearance of lactic acid from the body of man. When lactate is injected into a vein, equilibrium is gradually restored by elimination through the kidneys. The curve in Figure 2.2 shows the time course of this disposal process. It will be seen that a gradual return to a normal level is achieved by the organism. Another example is that of water ingestion following deprivation in the dog. Here the balance is restored over a short period of time, as is shown in Figure 2.3.

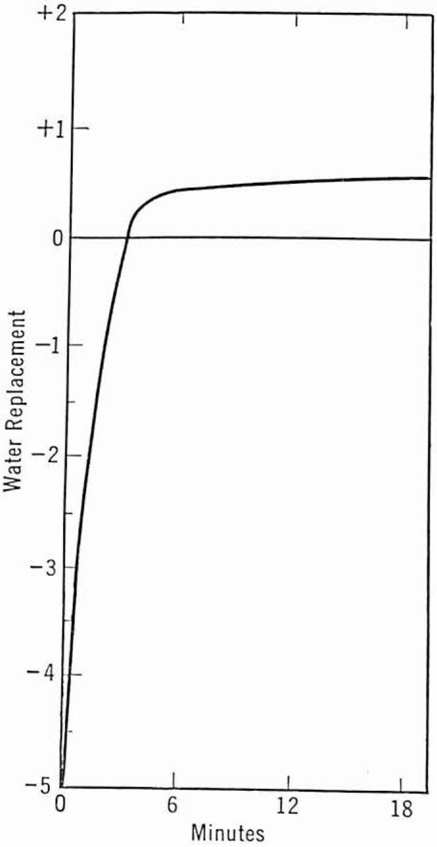


FIG. 2.3. Water replacement in the dog after deprivation. (After Adolph.[1])

Limitations of Homeostasis. Homeostasis is not perfect and organisms differ in their homeostatic mechanisms. Our previous example of the effect on a fish of increasing the saltiness of the water around it also implied that there is a limit to the organism's ability to maintain itself. Virtually any homeostatic mechanism has its limits. A man exercising in the hot sun will have a rise in internal temperature. The density of fats within the body can be changed by changing diet. Despite the tendency for blood volume to be restored when blood is lost, it is possible to bleed to death. Exploring the limits of various homeostatic mechanisms is an important task for physiology.

Homeostasis and Psychology. In the above lactic acid example, biochemical mechanisms were sufficient to restore balance and the adjustment is, therefore, almost entirely in the realm of physiology. But the water ingestion example involves more than this. To restore water balance, the organism must go into action, that is, behave, and we have arrived at a situation which often is discussed in elementary psychology under the heading of "thirst drive." Of course, nearly any physiological disequilibration might lead to behavior, even if the behavior were of the variety of drinking sodium bicarbonate or going to see a physician.

It is apparent, therefore, that the psychologist as well as the physiologist has a concern with the maintenance of physiological equilibrium. When physiological processes are insufficient to re-establish equilibrium, the total organism may be set into action and equilibrium restored behaviorally. This is especially characteristic of man, and behavior often intervenes even before the physiological processes have been called into great action. For example, shivering and constriction of the skin blood vessels will help maintain internal temperatures when the air is getting chilly, but so will putting on a coat, or building a fire, and these latter acts are likely to occur before the former have had much effect.

Man, in particular, may restore equilibrium in many different ways. Putting on a coat or building a fire illustrates overt manipulation of the environment at a very simple level, but some of our adjustments involve complex skills and the actual learning of these skills may be considered as an equilibratory process. Adjustment may also come through a reorganization of the way in which we regard the world, as might be exemplified by the results of successful psychotherapy. We say that the situation has acquired a new meaning for the patient, or that he perceives it differently, or that he is reoriented, or that he has accepted a new set of goals. This will, of course, result in changes in behavior, but is descriptively different from the taking-a-drink or putting-on-a-coat sort of adjustment. Although this kind of internal readjustment appears to be largely symbolic in character, it may involve the organism almost totally, including physiological adjustments within the **viscera.**

The maintenance of equilibrium is illustrated more fully in Chapter 20 on motivation. In the meantime, we should keep in mind that the organism may restore equilibrium by internal physical, chemical, and physiological changes, by reacting to change the external environment, or, especially in man, by undergoing a reorganization of thought processes which in turn modify overt behavior. Equilibrium is the end-product of the operation of certain mechanisms, not a teleological goal which works backward in time to determine bodily activities. Equilibrium is achieved when the organism possesses adequate internal mechanisms or receives adequate assistance.

The Integration of the Organism. The fact that the whole organism may appear to go into action under certain circumstances implies that some means of integration must exist. It is true, of course, that many physiological functions ordinarily involve in some way the entire organism. Indeed, one group of scientists adheres to a concept of "organismics" which implies the primacy of the organism-as-a-whole and which considers the activities of any part to be meaningless unless related to the whole. It is often pointed out that the properties of wholes cannot be predicted entirely from part properties alone. In chemistry, for example, the properties of water (H_2O) cannot be predicted from the properties of hydrogen (H_2) and oxygen (O_2) which combine to form it. Extreme forms of this view postulate supernatural organizing principles to account for organismic integration. Such an approach serves only to mask the extreme complexity of the part activities going on within the organism; it fails utterly to account for those cases where integration fails; and it is of little use in the practical situation where control of the activities of the organism, both internal and external, must be accomplished by manipulating the condition of a relatively small part of the organism.

It is more fruitful to consider neither part nor whole activities as being more real, but, rather, as scientific data, as depending upon the method of observation. It is possible to study the organism on any level of analysis which one wishes. The study of a single nerve fiber is neither more nor less laudable than the study of the behavior of the whole organism. The guiding principle here is that the level chosen depends on the problem at hand and cannot be arbitrarily decided in advance on the basis of a preconceived philosophical system. To take an extreme example, it is probably not necessary to examine a subject's legs for varicose veins when using him as a subject in an experiment on night-vision. On the other hand, in the same experiment, it would be very desirable to have some knowledge of the subject's over-all nutritional status. For any given situation, we must decide the purpose of the investigation and fit our analysis to that purpose. This point of view already has been partly expressed in the discussion of physiology and psychology, where the division between the two disciplines was found not to be a real or sharp one.

The Mechanisms of Integration. The integration of function in a single-celled animal, such as the amoeba, is a relatively simple process. Overt behavior is mediated largely through the properties of the cell membrane and internal functions are coordinated or integrated by direct biochemical activity in the cell itself.

Such an arrangement is not adequate for the integration of the activities of a complex organism, however, and higher organisms have developed two general ways in which integration is achieved. One is chemical and involves the transport of substances through the circulatory system. An example of this is the release of pancreatic fluid into the intestine as a response to a substance, secretin, that reaches the pancreas through the blood stream from the small intestine. Secretin is released from the intestine when acid food enters from the stomach. Here, a fairly complex integration has been shown to be largely chemical. Chemical changes within the body underlie many important activities (see Chap. 15) and certain changes may give rise to alterations of over-all activity of the variety which might be called personality.

The second great integrating system is the nervous system and this is the primary mechanism for coordinating rapid, precise activity. The various extensions of the nervous system reach virtually all parts of the organism and bring these parts into relationship with one another. Very dramatic failures of integration follow the separation of a part of the body from the bulk of the nervous system. If the nerve going to a given muscle is destroyed, as it might be in infantile paralysis (anterior poliomyelitis) , the muscle not only fails to respond as a part of the total organism, but also withers away. Or if the spinal cord is completely severed, there is no control of the portion of the body below the cut and integration fails. These examples demonstrate the dependence of the activities of the organism-as-a-whole upon specific connections among specific parts and make more intelligible the necessity for avoiding mystical concepts of organismic unity. They indicate, also, that we must know something about the nervous system if we are to achieve any understanding of organismic behavior.

Suggested Readings

1. Cannon, W. B., *The Wisdom of the Body*. New York: Norton, 1932.
2. Carlson, Anton J., and Johnson, Victor, *The Machinery of the Body*. Chicago: University of Chicago Press, 1953, Chap. 2.
3. Maier, N. R. F., and Schneirla, T. C., *Principles of Animal Psychology*. New York: McGraw-Hill, 1935, Chap. 1.

3 Conducting Cells and Nerve Action

We have considered the basic life processes found in single-celled organisms. Now we shall be concerned with one of those functions, conduction, as it occurs in multicellular organisms. In many-celled organisms, it will be remembered, the cells which form the organism show specialization for one or another function. The type of cell which is specialized for conduction is called a **neuron;** therefore, our task in this chapter is to consider the properties of neurons and the meaning of these properties for an understanding of behavior. Before considering neurons, however, it will be necessary to outline very briefly the evolution of the integrative mechanism and to introduce a few terms needed for later discussions.

The Development of Nervous Integration. Even single-celled organisms may contain specialized structures for conduction and contraction (Chap. 2). Paramecium is a good example of such an organism, since it contains conducting fibers within its cell contents, as well as **cilia,** which are highly specialized locomotor hairs on the surface. Possessing these structures, paramecium displays a promptness and precision of movement not possible for the less well-endowed amoeba. As the many-celled organisms evolved, total cells, rather than parts of cells, became specialized for these functions. The first specialized cells to evolve were contracting cells and are to be found in the sponges. These cells form rings of tissue around the pores and serve to close them when struck by, say, a piece of sand. Since these cells themselves perform the activity, they are called **effector cells** or **effectors.** In this case, the effector cells are specialized for contraction and are called muscle cells (*cf.* Chap. 5), but the general term "effector" is also applied to glands. The first step, therefore, is the development of effector cells which, because there are no specialized conductile cells, respond only when directly stimulated. For this reason the sponge can scarcely be called an integrated organism, since there is no conduction from place to place within the organism, and one small group of effector cells may respond to stimulation without affecting the activities of the rest of the animal. Obviously, an increase in efficiency would result from the development of conductile cells, so that the organism might respond in a truly integrated way.

This next step is partly taken in certain of the coelenterates, of which the sea anemone is a typical example. In this animal, the body

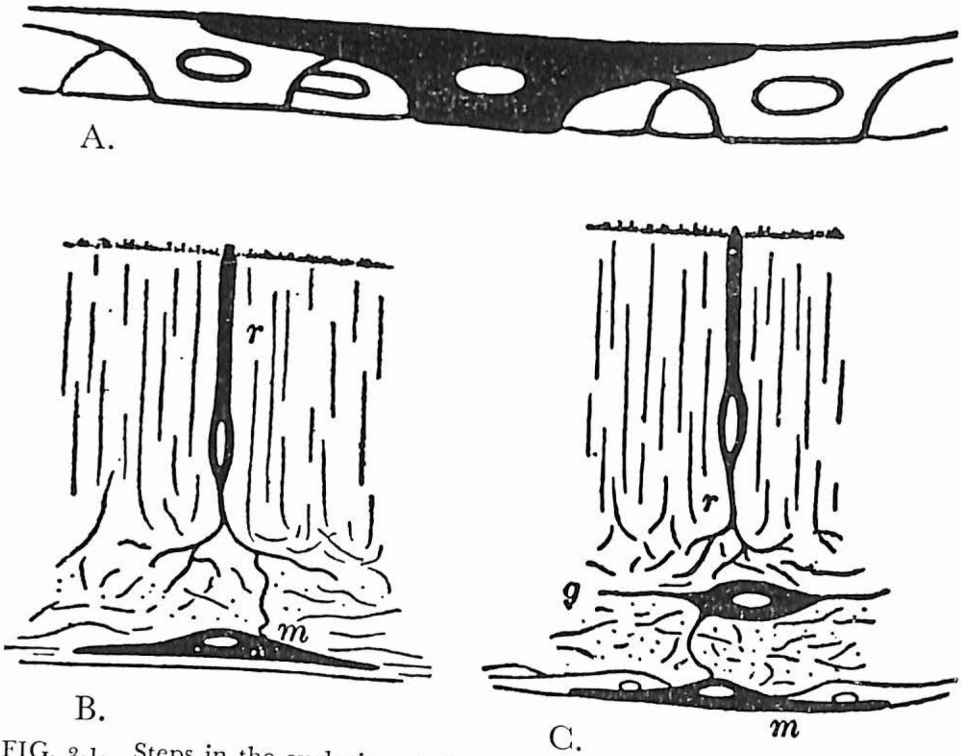


FIG. 3.1. Steps in the evolution of the receptor-effector relationship. The effector cell, A, evolved first, the combined receptor-connector cell, B, next, and the separate receptor and connector, C, appeared last. (From Parker.[1])

wall contains cells which are highly sensitive to contact and which send fiber processes to effector cells located between the body walls. Here a single cell is specialized for both irritability and conduction. A cell specialized for irritability is called a **receptor,** while one specialized for conduction is called a **connector;** thus the sea anemone possesses **receptor-connector** cells, since they are specialized for both functions.

The separation of receptor and connector cells has taken place in the jellyfish, which are also coelenterates. The jellyfish possess cells specialized for conduction only, called connector or **association cells,** that form a net-work which pervades every portion of the animal, with no concentration of nervous tissue at any particular point. Because of its appearance, such an arrangement is called a **nerve-net.** In the nerve-net, there is no polarity of conduction; that is, an impulse started at one point is conducted in all directions, despite the fact that the net is composed of separate neurons. In spite of this limitation, the swimming activity of the jellyfish, which involves the entire animal in an integrated manner, clearly demonstrates the tremendous importance of a specialized conducting system. These progressive steps — effector alone, effector plus receptor-connector, and finally receptor, connector, and effector — are illustrated in Figure 3.1.

Subsequent to the developments which are outlined above, the evolution of conducting tissues has involved relationships among cells, rather than changes in the conducting cells themselves. Whereas it is characteristic of a nerve-net that there is no accumulation of cells in any one region, higher organisms have nervous "centers," that is, accumulations of interrelated neurons which serve to integrate the activities of the organism. We shall be concerned in Chapter 4 with this latter aspect of nervous evolution.

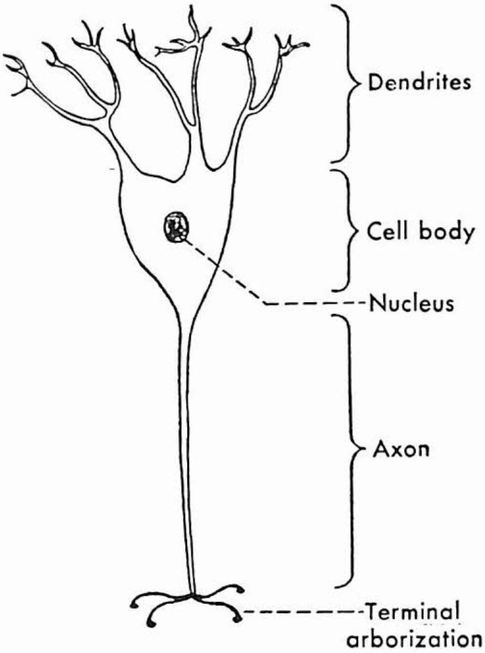


FIG. 3.2. Diagram of a "typical" neuron, showing the main parts.

The Neuron: Primary Structure. A neuron is shown diagrammed in Figure 3.2 and is seen to consist of three major subdivisions: one or more **dendrites,** a **cell body,** and one **axon.** The most prominent difference between an unspecialized cell and a neuron is the possession by the neuron of the fiber processes (dendrites and axon), which permit a single cell to extend over a considerable distance. In a tall man, for example, a single neuron may be over 6 feet in length, extending all the way from the big toe to the base of the skull. Despite this length, neurons are microscopic in diameter. Cell bodies range between 4 and 135 microns in diameter (a micron is one thousandth of a millimeter), while axons are between 1 and 20 microns in diameter. Of course, most neurons are shorter than the ones referred to above and many of them extend less than a millimeter.

In addition to the typical neuron diagrammed in Figure 3.2, there are many other types, some of which are shown in Figure 3.3. Neurons are often classified according to the number of fiber processes on them; thus neuron " A " in Figure 3.3 is called **unipolar** because there is only one process, neuron " B " is called **bipolar,** and neuron " C " is called **multipolar.**

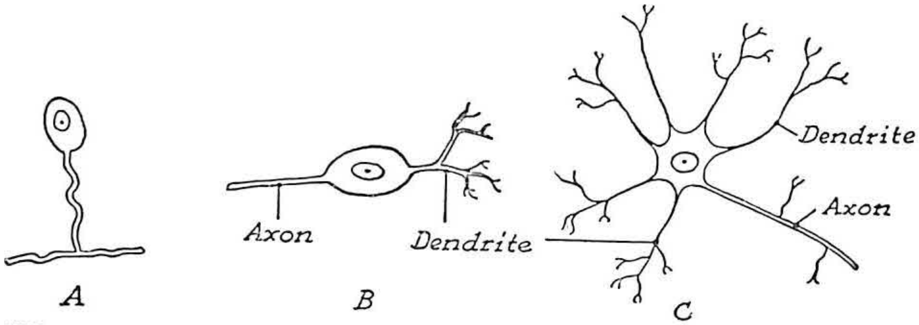


FIG. 3.3. Three types of neuron: (A) monopolar, (B) bipolar, (C) multipolar. (From Buchanan.[2])

The Neuron: Accessory Structures. Neurons are associated with accessory tissues which serve to support and nourish them, the particular structures depending upon the location of the neuron. Outside the **central nervous system** (the brain and spinal cord), neurons are ordinarily found in **nerve trunks,** or more simply termed, **nerves.** The bulk of these **peripheral** nerves (" peripheral " in this case means outside the central nervous system) is supporting tissue rather than active nervous tissue; one should distinguish carefully between the term *nerve,* which refers to the total structure including both supportive and neural tissue, and the term "**nerve fiber,**" which refers to the fiber process of a single neuron. Figure 3.4 shows a cross-section of a nerve.

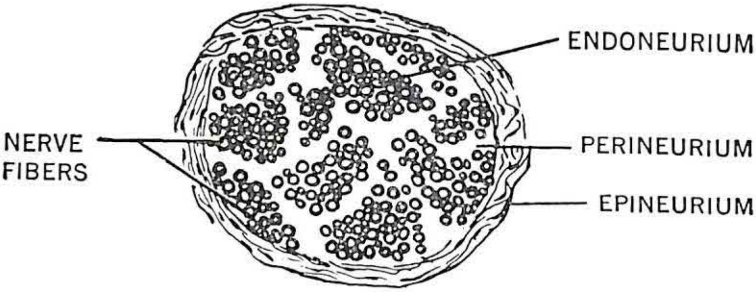


FIG. 3.4. Cross section of a nerve, showing the separate nerve fibers which are contained in it. (From Best and Taylor.[3])

Closely adherent to some but not all nerve fibers may be found either one or both of two accessory structures, the **myelin sheath** and the **neurilemma.** The myelin sheath is shown in Figure 3.5 and consists primarily of a white fatty material which gives the characteristic appearance to the so-called **white matter** of the central nervous system. The **gray matter** consists of cell bodies and those nerve fibers that are unmyelinated. Myelin is sometimes thought to act as an insulator and reduce the effect of adjacent nerve fibers upon each other.

The neurilemma is a thin layer of living cells which may surround peripheral but not central nerve fibers. When we discuss the regeneration of cut or crushed nerve fibers, the importance of this distinction between central and peripheral nerve fibers will become apparent. If we consider all of the possible combinations of neurilemma and myelin, we find that there are four — neurilemma alone, myeline alone, both present, and both absent. All of these possible combinations occur: central fibers may or may not be myelinated, but never have neurilemma, while peripheral fibers may or may not be myelinated, but characteristically have neurilemma.

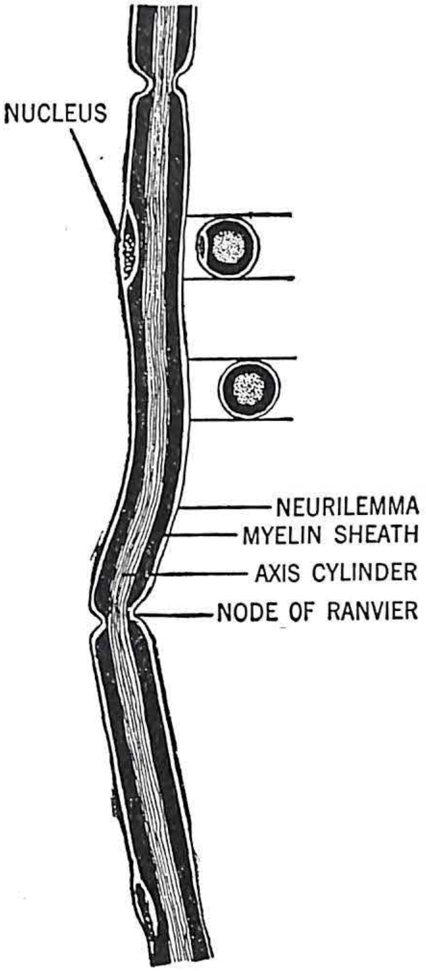


FIG. 3.5. A motor neuron showing the principle parts. The " nucleus " in the diagram refers to the nucleus of a neurilemma cell, not of the neuron. (From Best and Taylor.[4])

The Neuron: Regeneration. An animal, such as man, has at birth its full complement of neurons and no new ones are subsequently produced. This inability of neurons to reproduce themselves is shared with other highly specialized cells, such as muscle cells. It contrasts with the ability of some cells, e.g., those of the skin, to produce more cells when some are destroyed (when a cut heals, for example), or to produce new cells continually. It follows that if the cell body of a neuron is destroyed, no replacement of the loss can ever take place. This is the case with anterior poliomyelitis (infantile paralysis) which destroys the cell bodies for the neurons which send axons to skeletal muscles.) Any improvement in function, such as might result from exercise in warm water, must arise from the strengthening of those muscle cells still served by neurons and not by the restoration of lost neurons.

(If a nerve fiber is crushed, cut, or otherwise damaged, while the cell body remains intact, the situation sometimes is more hopeful.) Under these circumstances most nerve fibers which are provided with neurilemma will regenerate; therefore, most peripheral nerve fibers will regenerate but central fibers, which always lack neurilemma, will not. The restoration of function depends upon whether or not the regenerating fibers grow back through the old neurilemma tubes of the cut nerve, where the cut axons have degenerated. This is ordinarily accomplished by sewing the nerve together where it has been severed so that the regenerating fiber will eventually reach the muscles and receptors formerly served by the nerve. This is a fairly slow process, since about 1 millimeter per day is a fair average for the regrowth of a cut fiber.

(**The Neuron: Embryonic Origin.** It is now generally agreed that all neurons in vertebrate organisms arise from the **neural tube,** which forms from the outermost layer of cells in the embryo. As the embryo grows the sensory and motor neurons elongate and establish connections with receptors and effectors, and association neurons form the pathways which lie within the central nervous system, or CNS, itself. It is not entirely clear why a given neuron should grow in a particular direction rather than in another, but we may assume that the direction of growth is determined by the biochemical environment of the growing fiber. Attempts have been made to explain learning in terms of neuron growth and we shall have occasion to consider this topic in a later chapter.)

The Nerve Impulse: Introduction. It has been known for some time that the nerve cells are specialized for conduction, but only a little over 100 years ago it was generally thought that the impulse was so rapid as to prevent measurement. After Helmholtz had measured the speed of conduction in both frog (about 7 meters per second) and man (120 meters per second maximum), it became obvious that the nerve impulse is comparable neither to the conduction of light and electricity (300,000,000 meters per second), nor of sound (331 meters per second in air). Even the fastest impulses in man are slow compared to the speed of modern airplanes and many of us could run faster than the slowest impulses (about 0.7 meters per second). Although these speeds are sufficient reason in themselves for giving up any attempt to explain conduction in terms of electrical circuits, as in a wire, there is now considerable evidence which suggests a different type of explanation and leads to the conclusion that the nerve impulse is propagated by the progressive spread of electrochemical changes localized primarily in the cell membrane.

The Nerve Impulse: Electrical Evidence. Most research upon the nerve impulse is now accomplished by recording the electrical changes occurring as an impulse passes. In order to understand the meaning of the results so obtained, it is necessary to have a clear conception of what is being recorded.

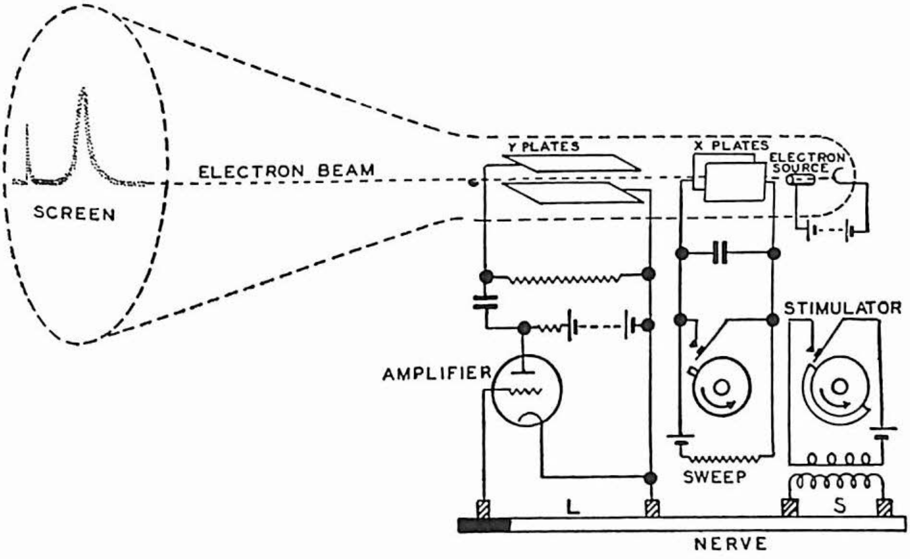


FIG. 3.6. Cathode ray oscilloscope used as a sensitive galvanometer for recording nerve potentials. The potential changes are amplified and appear on the screen of the cathode ray tube as changes in the elevation of a rapidly sweeping spot. (From Erlanger and Gasser.[5])

Fundamentally, the electrical method depends on the fact that an active tissue will show an electrical potential different from that of an inactive one. If we attach a delicate meter, designed to measure very small changes in electrical potential (voltage), to a resting nerve fiber it will be found that all points along the fiber are neutral to one another. If the fiber is excited, the point which is active at a given moment, that is, which the impulse is passing, will be negative as compared to the rest of the fiber. Figure 3.6 shows diagrammatically such an apparatus. Only one electrode need be attached to the fiber, and the other may be grounded elsewhere on the organism being studied. The potential-recording device, or galvanometer, is the critical part of the apparatus and only the development of modern electronic devices has made possible the full realization of the potentialities of the method.

Figure 3.7 illustrates a typical potential curve from a single nerve fiber conducting an impulse. One may identify the **spike potential,** the **negative after-potential,** and the **positive after-potential.** The relative duration of each of these three sections of the curve has been found to depend upon many factors, such as the chemical concentration of the surrounding medium and the extent of previous activity.

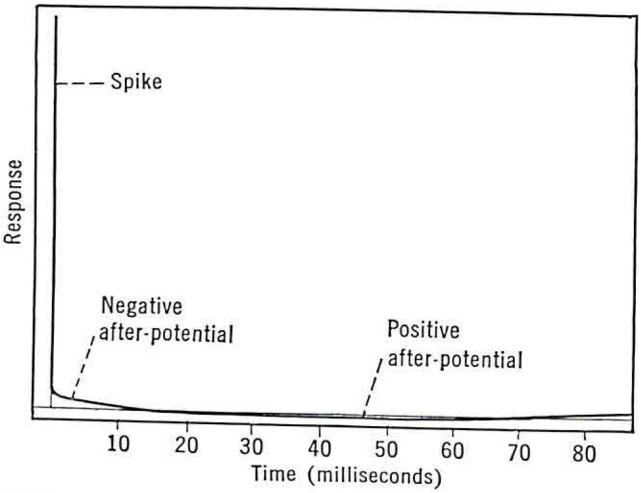


FIG. 3.7. Action potential of a " A " type fiber. (From Gasser.[6])

Closely related to the potential changes will be found changes in the excitability of the fiber. If the **threshold** of **excitability,** i.e., the lowest energy that will elicit a nerve impulse, is measured by means of electrical stimulation, it is possible to draw a curve of excitability. Such a curve is shown in Figure 3.8. During the first portion of the spike potential no current, no matter how strong, will excite the fiber and this period is called the **absolute refractory period.** Its duration is 1 to 10 milliseconds, depending on the particular fiber, and thus the maximum number of times that a given fiber can be excited in a second will range from 100 to 1000. During the remainder of the spike potential, the fiber may be excited only by a suprathreshold shock. This is called the **relative refractory period** and is two to three times as long as the absolute refractory period. Following this there is a period of superexcitability for the duration of the negative after-potential, which may last several times as long as the relative retractory period and during which shocks of somewhat subthreshold magnitude may elicit a nerve impulse. Finally, a prolonged subnormal excitability period occurs during the positive after-potential.

These changes in excitability associated with the passage of nerve impulses have considerable importance in our understanding of nervous function. Not only must they be accounted for by any satisfactory theory of conduction, but, also, they impose certain limitations on hypotheses concerning nervous system functioning which involve the action of nerve fibers. One example will suffice. An early theory to account for the perception of pitch suggested that each fiber in the auditory nerve carried impulses with the same frequency as the stimulating sound wave. This would require (Chap. 11) that each fiber should be capable of carrying impulses at the rate of 20,000 each second. But this is impossible. If the most rapidly recovering fibers require a millisecond to pass out of the absolute refractory period, they cannot be stimulated more often than once every 1/1000 second, or 1000 times per second. Thus, the theory fails because it requires nerve fiber functioning which we know to be impossible.

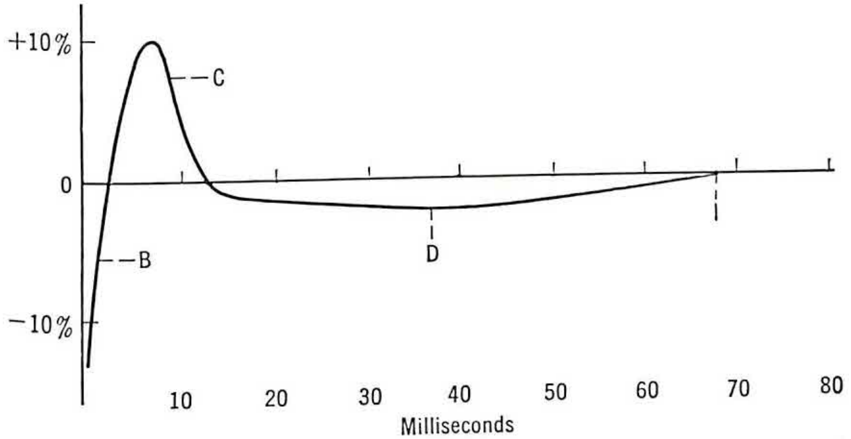


FIG. 3.8. Changes in excitability of a nerve fiber after the passage of an impulse, expressed as percent of original excitability. B is the relative refractory period, C the supernormal period, and D the prolonged subnormal period. The absolute refractory period immediately precedes B. (From Gasser.[7])

The Nerve Impulse: Chemical Events. Although the local chemical changes, which must be taking place during the passage of a nervous impulse, cannot be observed directly, it is possible to demonstrate the utilization of oxygen by active nerves, the release of carbon dioxide, and the production of minute amounts of heat. As is the case with muscles, peripheral nerves are able to run an **oxygen debt,** that is, they will conduct impulses for a time in the absence of oxygen and then consume extra amounts after the oxygen is restored. This would indicate that oxygen is utilized during the recovery phase, rather than in the conduction of the impulse, and this will be seen to be compatible with the discussion of the nerve impulse in a later section. Central nervous tissue, however, cannot accumulate an oxygen debt and, therefore, is very vulnerable to asphyxiation. For example, infants who become entangled with the umbilical cord during delivery, and, therefore, are without an oxygenated blood supply for an extended period of time, may show destruction of central nervous tissue and a consequent reduction in intelligence. Fortunately, infants are very much more resistant to asphyxiation than adults, so that the risk is not as great as might be expected. The extra sensitivity of the central nervous system (CNS) to oxygen lack is due to the higher metabolic rates of the cell bodies and structures at junctions between neurons. The CNS fibers are similar to peripheral nerve fibers in their metabolism.[1]

The exact chemical events during the passage of and recovery from the nerve impulse are not known. No doubt certain complex phosphorus compounds such as phosphocreatine are involved since the impulse is associated with a breakdown of this substance and since it is resynthesized during the recovery period. Also, radioactive phosphorus has been found to diffuse more rapidly into active than into inactive fibers. The energy source in peripheral fibers is not completely known, but we may be confident that the activity of the brain is maintained primarily by the oxidation of **glucose,** or blood sugar. The behavioral symptoms, ranging from lassitude to coma, which may result from an abnormally low blood sugar level serve to emphasize the importance of understanding the chemical basis of nervous activity.

The Excitation of Nerve Fibers. The magnitude of a nerve impulse does not depend upon the magnitude of the stimulus initiating it. This fact is formalized by the so-called **all-or-nothing law,** which states that a fiber responds to the limit of its capabilities, or not at all. The stimulating energy must exceed a certain value, or threshold, in order to excite the fiber, depending upon the particular fiber involved. A fiber may be stimulated by many kinds of stimuli, among them pressure, chemicals, and electricity; but in quantitative research the use of electricity is universal, since mild electric currents do not injure the tissues and precise control of intensity and duration is possible.

The effect of an electrical stimulus on a nerve fiber depends upon the polarity of the current. The cathode (negative) electrode is stimulating, the anode (positive) is not. If a brief shock is applied, too weak to initiate a nerve impulse, a **local excitatory process** may be observed. This consists of a shift in membrane potential confined to the region of the stimulating electrode. This process may actually increase after the stimulus has ceased, indicating that it is an active response of the fiber. If the local excitatory process is of sufficient magnitude, a propagated disturbance, the nerve impulse, results. The magnitude of the resulting impulse is not affected by the magnitude of the local excitatory process, once the threshold has been exceeded. The magnitude of the local excitatory process, however, is related to the strength and duration of the stimulating current.

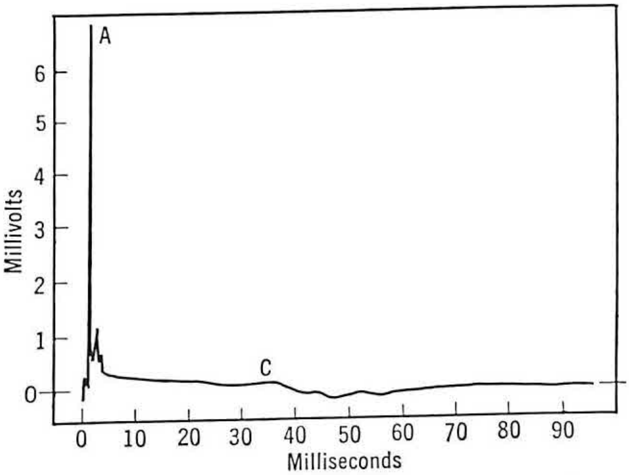


FIG. 3.9. Showing the A and C components in a mixed nerve potential (there is no B component in this example). (From Gasser.[9])

Summation of the effects of two rapidly successive brief subthreshold shocks can be accounted for by the persistence of the local excitatory process. The effect of the second shock sums with the local excitatory process invoked by the first stimulus and the threshold of the nerve impulse may then be exceeded.

The Speed of the Nerve Impulse. As mentioned earlier, the fastest nerve impulses in men travel at a rather modest pace, specifically, about 120 meters per second. It must not be imagined, however, that all nerve fibers conduct at this rate. If a peripheral nerve is stimulated and the resulting impulses recorded at a distance from the point of stimulation, it will be found that a wide curve of potential change will be recorded. This curve, an example of which will be found in Figure 3.9, represents the summation of the individual potential changes attributable to the impulses in individual fibers. The wide extent in time shown by the total curve must indicate that the various nerve impulses started along individual fibers of the nerve by the stimulus have not all reached the recording point at the same time and, therefore, have traveled at different rates. Careful measurement reveals that the range of speed is quite great, the fastest impulses traveling at 120 meters per second, the slowest at 0.7 meters per second. It may further be observed that it is possible to group the impulses and, hence, the nerve fibers responsible for them into three large groups. These are called A, B, and C, with type A fibers conducting most rapidly, and type C most slowly. Table 3-1 has been worked out to summarize some of the properties of these different types.

Table 3-1. Properties of A, B, and C Fibers of Peripheral Nerves

[Modified from Brink.[10]]

	A	B	C
Diameter, in microns	20–1	Less than 3	Any unmyelinated
Conduction speed, meters per sec.	100 to less than 5	14 to less than 3	less than 2
Spike duration, milliseconds	0.4–0.5	1.2	2.0
Negative after-potential duration in milliseconds	12–20	None	50–80
Positive after-potential duration in milliseconds	40–60	100–300	300 to more than 1000

The Processes in the Nerve Impulse. We have considered the electrical changes observable as an impulse passes along a nerve fiber and have said something about the grosser chemical changes. We must now consider in more detail what happens in a single fiber as the impulse sweeps along it. As implied by the all-or-nothing law, the conduction of an impulse is a function of the fiber itself — it supplies the energy for conduction, much as the energy for the passing of a fire along a fuse is dependent upon the burning of the material of the fuse itself. In general, there are three things which need accounting for: (1) the changes occurring in the nerve fiber as an impulse passes; (2) the manner in which an active section of fiber excites the next region, so that the impulse travels, or is propagated; and (3) the processes involved in recovery.

The most generally accepted view is that conduction depends upon the cell membrane. The nerve fiber membrane displays what is known as a membrane potential. If we measure the electrical potential across the membrane, an operation which can be performed directly in the giant axon of the squid, we find the outside is positive in relation to the inside of the cell. In a typical neuron, this potential is about 50/1000 volt. If our measurement is continued as the neuron is stimulated, we find that as each impulse passes this potential is reversed momentarily — in fact, we shall observe the same electrical changes that we have previously discussed. Apparently, therefore, we can account for the electrical phenomena of the nerve impulse by assuming them to be the result of changes in the polarization of the cell membrane. Our next task is the filling in of the important details.

The membrane potential in the resting neuron is maintained by what has been called the "sodium pump." Some chemical mechanism, requiring oxygen, constantly pumps sodium ions outward through the membrane. Since sodium ions are positively charged, the outside of the membrane becomes positive in relation to the inside. The nerve impulse consists in a breakdown of this polarization, with a sudden flow of sodium ions toward the inside of the membrane and, incidentally, a leakage of potassium ions from the fiber. The membrane is rapidly re-established in its polarized state, the period of complete potential reversal corresponding to the absolute refractory period.

The passage of the impulse along a fiber involves the propagation of the breakdown of polarization. This is probably accomplished by local flows of charged particles (ions), in other words, by local electrical currents. Figure 3.10 shows how this is accomplished. At a given instant, the impulse is at a given place along the membrane. This place is negative on the outside in relation to the adjacent membrane. As a result, there is a local flow of current as indicated by the little arrows and this is apparently sufficient to set into action chemical events which lead to the breakdown of the membrane potential in the next region of the membrane, and so on and on along the fiber. Immediately after the passage of the impulse, the chemical structure of the membrane is re-established, the sodium pump returns to action, and the membrane potential is reconstituted. No doubt this requires chemical work and involves oxidation, but the details have not been worked out.

Stimulation of a fiber involves the local reduction of the membrane potential to a critical point, the threshold. If the reduction is less than threshold, no impulse results, although two such subthreshold stimuli can summate, as we have seen in a previous paragraph. The local excitatory state is thus explained by membrane theory as a subthreshold local shift in the membrane potential. Since such a shift tends to persist for an instant, and since it also tends to spread a short distance along the fiber, both temporal and spatial summation are readily accounted for.

Nervous Fatigue. Although it is often stated that nerve fibers do not show fatigue, this is not strictly true. The refractoriness of nerve fibers after the passage of each impulse limits the number of impulses which may be initiated per second and, thus, at least a minimum of recovery time is assured. The longer continued and the more frequent the stimulation, the more slowly a fiber will recover and, therefore, the fewer impulses per second it will carry. In the intact organism, however, other structures associated with action will fatigue sooner than nervous tissue, so nervous fatigue is not the controlling factor. The student is also cautioned against thinking that the " mental fatigue " which follows, for example, a bout of studying calculus is directly and primarily dependent upon the fatigue of individual nerve fibers in the brain. Such is almost certainly not the case.

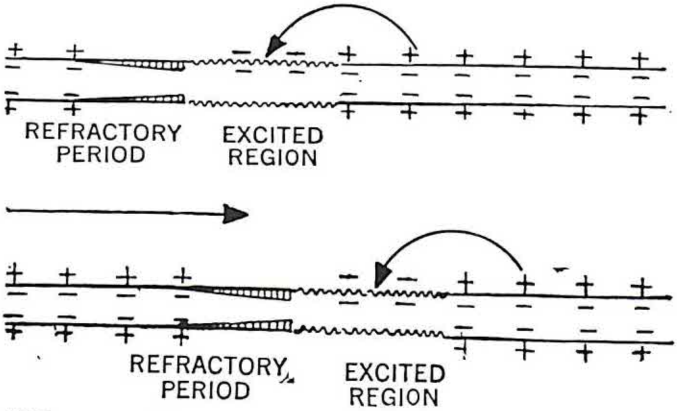


FIG. 3.10. The membrane potential in a longitudinal section of a nerve fiber. As the impulse passes, the membrane becomes completely permeable, and there is a local flow of current as indicated by the arrow (it is conventional in neurophysiology to indicate flow from positive to negative). The potential is reconstituted after the impulse has passed. (From Best and Taylor.[11])

The Synapse. In a nerve-net there may be continuity of tissue from neuron to neuron. In the more complex animals such as man there is little doubt that neurons are separate. It is apparent that if one neuron is to cause another to conduct a nerve impulse there must be some point of functional contact between them. Such a point of functional contact is called a **synapse.**

Synapses ordinarily occur between the axon of a neuron and either the cell body or dendrites of another, but it is an over-simplification to think of this relationship as involving one branch of one axon and one cell body or dendrite, much as one telephone wire connects to the next. In fact, the *terminal arborization* of a given axon provides many branches which may end upon a number of adjacent neurons, or provide several terminations on a single neuron. Figure 3.11 shows the cell body and parts of the dendrites of a cell from the spinal cord, and it will be seen that there are very many synapses on even the one cell. In some cases, as in Figure 3.12, the axons of many fibers may entwine with the dendrites of others, forming a dense mass of fibers.

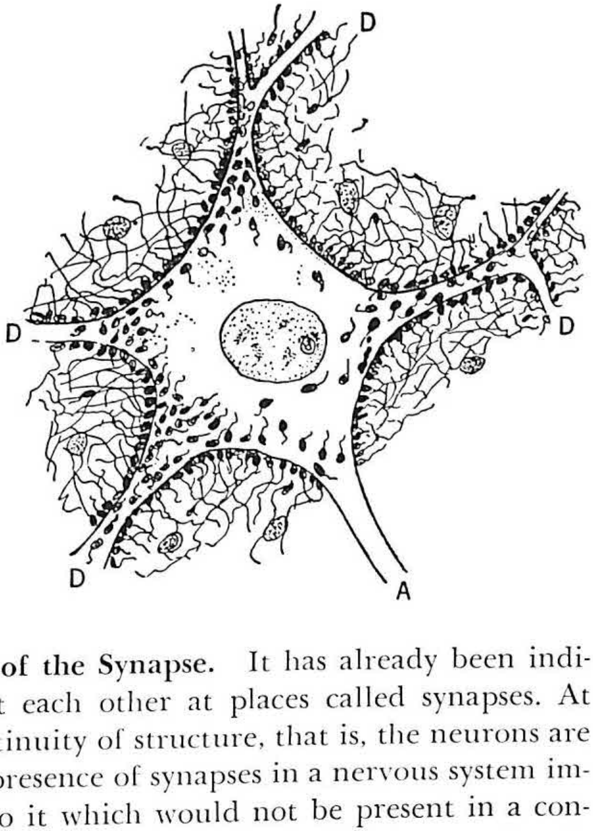


FIG. 3.11. Synapses on the cell body and dendrites of a motor neuron in the spinal cord. (From Best and Taylor.[12])

Functional Properties of the Synapse. It has already been indicated that neurons affect each other at places called synapses. At synapses there is a discontinuity of structure, that is, the neurons are separate structures. The presence of synapses in a nervous system imparts certain properties to it which would not be present in a continuous system. One of these synaptic properties is **synaptic delay.** It requires slightly more time for an impulse to pass across a synapse than to run along an uninterrupted fiber. Another property is increased susceptibility to drug action. This probably results from the absence of myelin near the synapse, which permits readier access of drugs to the axon.

The most important property of the synapse from the point of view of integrated activity is its characteristic one-way conduction, so that impulses may pass from one axon to the next dendrite or cell body, but not in the opposite direction. It is the synapse, therefore, which imparts the **dromic** character to nervous activity, that is, establishes the normal direction of conduction. This property is not shown by individual nerve fibers, which will conduct **antidromically** as well as **dromically.** So the interposition of synapses between neurons is of the utmost importance.

Since the structure of one neuron is not continuous with the next, it is necessary to consider the manner in which an impulse in one can initiate an impulse in the second. Two general theories have been proposed, one of which states that transmission across a synapse is basically electrical in nature. When the wave of negativity which is part of the nerve impulse reaches an axon terminal, it is thought that the potential difference then existing across the synapse is sufficient to depolarize the membrane of the subsequent neuron and, therefore, give rise to a nerve impulse in this second neuron.

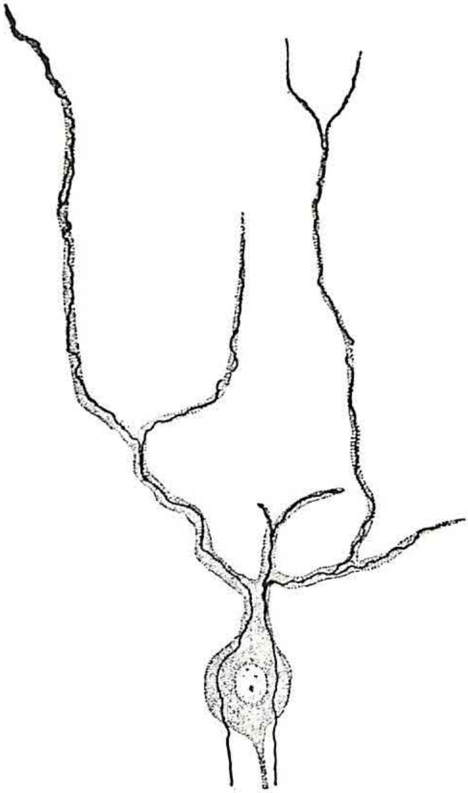


FIG. 3.12. Intertwining nerve fibers in the cerebellum. (From Kuntz.[13])

The second theory proposes that transmission across a synapse is primarily chemical in nature. Nervous activity of at least some synapses involves the secretion of a substance known as acetylcholine. This leads to the suggestion that acetylcholine may provide the means by which one neuron causes the next to discharge. The axon of one neuron is supposed to secrete this substance, which then leads to the discharge of the next neuron. The liberated acetylcholine is removed very rapidly by the enzyme **cholinesterase.** It appears that chemical mediation is indeed the case in certain peripheral synapses, but considerable dispute has arisen concerning the correct interpretation for most of them.

In any case, it is exceedingly unlikely that a single impulse will cross a synapse. **Summation** will be discussed later in greater detail. It is well to note here, however, that the complexity of even the most simple types of connection results in part from the necessity of precise timing of impulses in order to permit conduction to the post-synaptic neuron.

It should be noted that synaptic relationships may show one of two varieties of functional influence, **excitation** or **inhibition.** Inhibition (i.e., reduction or elimination of response) is not solely a synaptic property; but some synapses produce an inhibition of the post-synaptic neuron rather than its excitation. The importance of this point will become evident in Chapter 16.

Effects of Parallel Fibers. Although the influence of one neuron on another is most obviously exercised through synapses, it is likely that nerve fibers lying parallel to each other in, for example, a nerve trunk have some effect on each other. Since the nerve impulse involves an **electrical field,** it is not surprising that the passage of an impulse in one fiber should change the excitability of other adjacent fibers.[2] Where neurons are tightly packed, as they are in the central nervous system, it is possible that the discharge of groups of neurons synchronously may depend on some such mechanism. As will be seen later (Chap. 19), we have to take account of the effects of adjacent fibers on each other when we consider the theoretical possibilities of brain action.

The Neuron and Behavior. Understanding the functioning of the neuron is basic for the development of an adequate account of organismic functioning. On the theoretical level, since it is the functioning of neurons which underlies all nervous integration, a complete physiological psychology should explain how the billions of neurons, each obeying the all-or-nothing law, combine to give the smooth, continuous adjustment characteristics of complex living organisms. Furthermore, and of practical importance, knowing how various conditions affect the functioning of neurons permits us to predict the effect on total behavior of these same conditions. When we consider the action of the central nervous system we shall again consider the problem of the neuronal basis of behavior.

Suggested Readings

1. Brink, Frank, Jr., "Excitation and Conduction," Stevens, S. S. (Ed.). *Handbook of Experimental Psychology.* New York: Wiley, 1951, Chap. 2.
2. Brink, Frank, Jr., "Synaptic Mechanisms," Stevens, S. S. (Ed.). *Handbook of Experimental Psychology.* New York: Wiley, 1951, Chap. 3.
3. Erlanger, J., and Gasser, H. S., *Electrical Signs of Nervous Activity.* Philadelphia: University of Pennsylvania Press, 1937.

4 | Introduction to the Central Nervous System

In the preceding chapter we defined the central nervous system (CNS) as the brain and the spinal cord. The term " central " is an extremely apt one, since there is no function of the normal adult human which is not influenced by the action of the CNS and which does not in its turn influence the action of the CNS. The CNS is, in a very real sense, the integration center of organismic function. As evidence of the truth of this point one may cite the growing body of neurophysiological results which indicate the intimate relationship between the CNS and bodily activities. As a practical example, one may cite the field of psychosomatic medicine, which is based upon the fact that many, perhaps all, bodily processes, e.g., digestion, the formation of skin rashes, and heart action, may be influenced by thoughts and anxieties, that is, by central nervous patterns.

It will be well to emphasize at once that the CNS does not fulfill this important role in integration by virtue of any mysterious or unique property. The basic property of central neurons is the same as that of peripheral neurons, that is, conduction. But the neurons of the CNS are interrelated in extraordinarily complex ways and there are so many of them that it is not possible for the student or the neurologist to understand the action of the CNS in terms of the contribution of each separate neuron, except in very general terms. Fortunately for our discussion, the central neurons group themselves into various structures which seem to have rather consistent functions. It is possible, therefore, to discuss the functions of a particular part of the brain rather than of the myriad neurons which form the elements of that structure. Nevertheless, the ultimate anatomical unit is still the neuron, and central neurons conduct nerve impulses which are essentially the same as those in peripheral neurons.

Thus, it is readily apparent that the functions of the CNS must depend largely upon its relationships to other bodily structures and upon interrelationships among its own subdivisions. The CNS has the properties of an amazingly complex control box provided with input circuits, output circuits, and many internal links. The input circuits are the afferent neurons, feeding messages into the CNS as the result of energy changes both in the external environment and within the tissues of the organism itself. The output circuits are the efferent fibers, leading impulses from the CNS to the effector mechanisms, the muscles, and glands. And of great importance are the internal links, the association neurons within the CNS itself, which serve to sustain and correlate the activities of the entire organism.

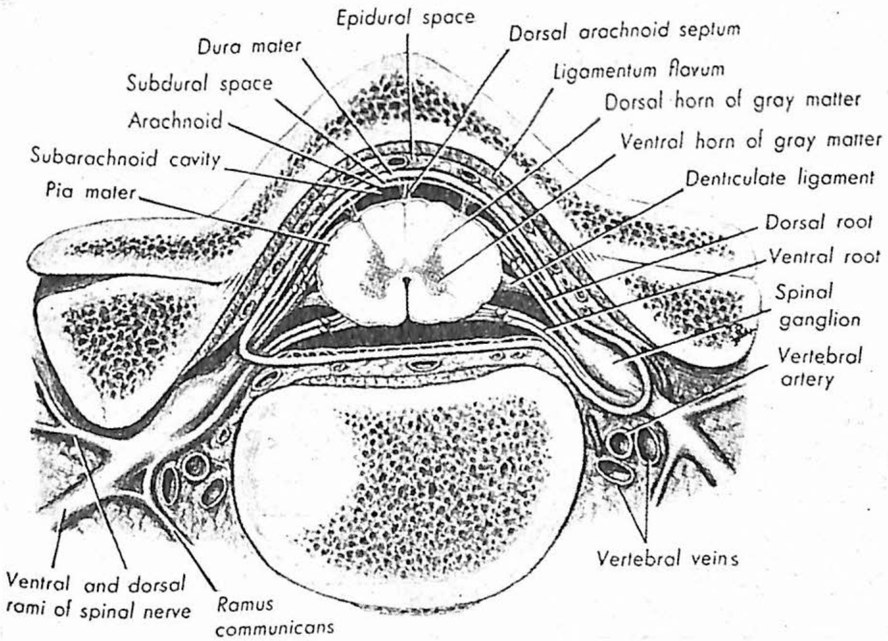


FIG. 4.1. Cross section of the spinal cord lying in place in the spine. (From Smith and Copenhaver.[2])

BASIC STRUCTURE OF THE CNS

The frontispiece shows a middle line section of the skull with the CNS in place. It will be noticed that the CNS lies within the skull and three membranes, or **meninges** (sing. *menix*). Starting with the one nearest the bone, these are called the **dura mater, arachnoid,** and **pia mater.** They are important in supporting the CNS, which is actually very soft, and they also enclose the cerebrospinal fluid, which bathes the entire CNS. The meninges and cerebrospinal fluid do not have conducting or integrating functions of their own, but rather serve to support and nourish the cells of the CNS itself. It will be seen from the figure that the junction between spinal cord and brain occurs approximately at the base of the skull. This boundary is not a sharp one and the distinction between brain and spinal cord is somewhat arbitrary.

In very primitive ancestors of the vertebrates and in embryos of all vertebrates, including those of man, the CNS is basically a tube lying near the surface of the back. All of the evolutionary elaboration which occurs never completely obscures this origin, and the adult CNS is hollow. In the spinal cord this opening is almost obliterated, but in the brain it persists as several large chambers called **ventricles,** all filled with cerebrospinal fluid. In cases of **hydrocephaly,** often seen in institutions for the feeble-minded, these ventricles become much enlarged and the surrounding tissues are severely damaged, resulting in great disturbances in behavior.

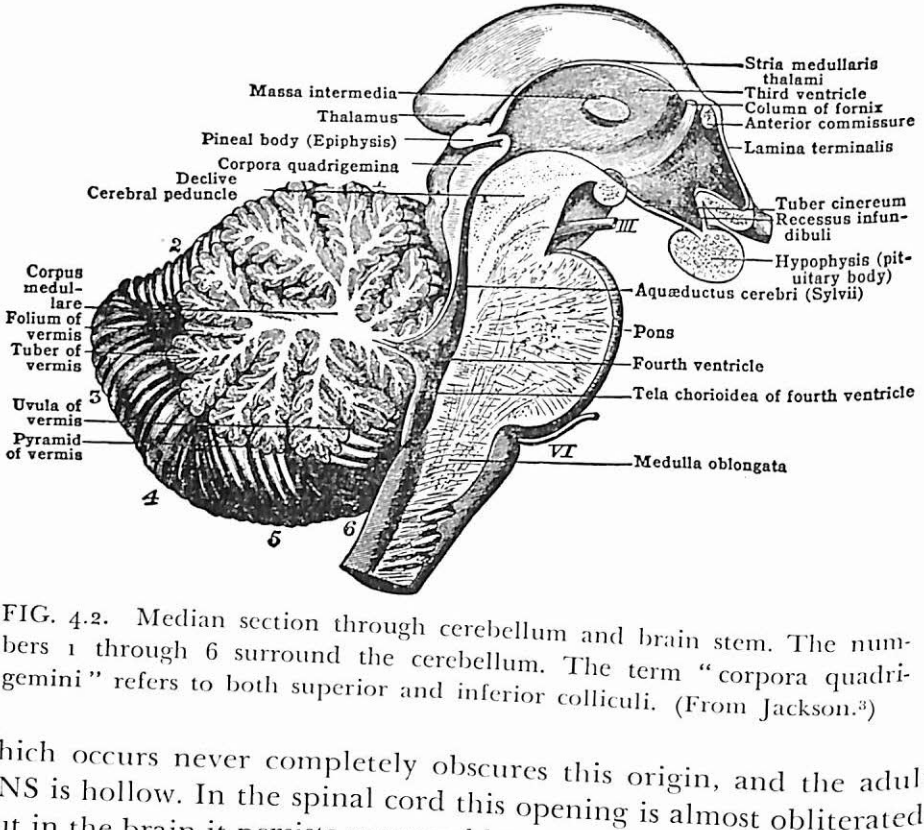


FIG. 4.2. Median section through cerebellum and brain stem. The numbers 1 through 6 surround the cerebellum. The term "corpora quadrigemini" refers to both superior and inferior colliculi. (From Jackson.[3])

The Spinal Cord. The cross section of the spinal cord found in Figure 4.1 shows its characteristic structure, with columns of white matter on the outside, the "butterfly" of gray matter on the inside. The white matter consists of conducting tracts of myelinated nerve fibers. The gray matter consists of cell bodies and unmyelinated fiber terminations. This arrangement is basic in the CNS and only in a few places is it reversed so that the gray matter lies to the outside. Such a place is called a **cortex,** and will be described in a later paragraph.

The spinal cord is connected to the periphery through 31 pairs of spinal nerves. Each spinal nerve (with one exception which is not of importance at the moment) joins the spinal cord through two roots, a **dorsal root,** which may be identified by the swelling to be seen on it (the **dorsal root ganglion**), and a **ventral root.** With only a few exceptions, the fibers in the dorsal roots are afferent and have their cell bodies in the dorsal root ganglia. The ventral roots are efferent and contain fibers which have their cell bodies within the gray matter of the cord. Injury to a dorsal root will affect sensation, while injury to a ventral root will affect movement.

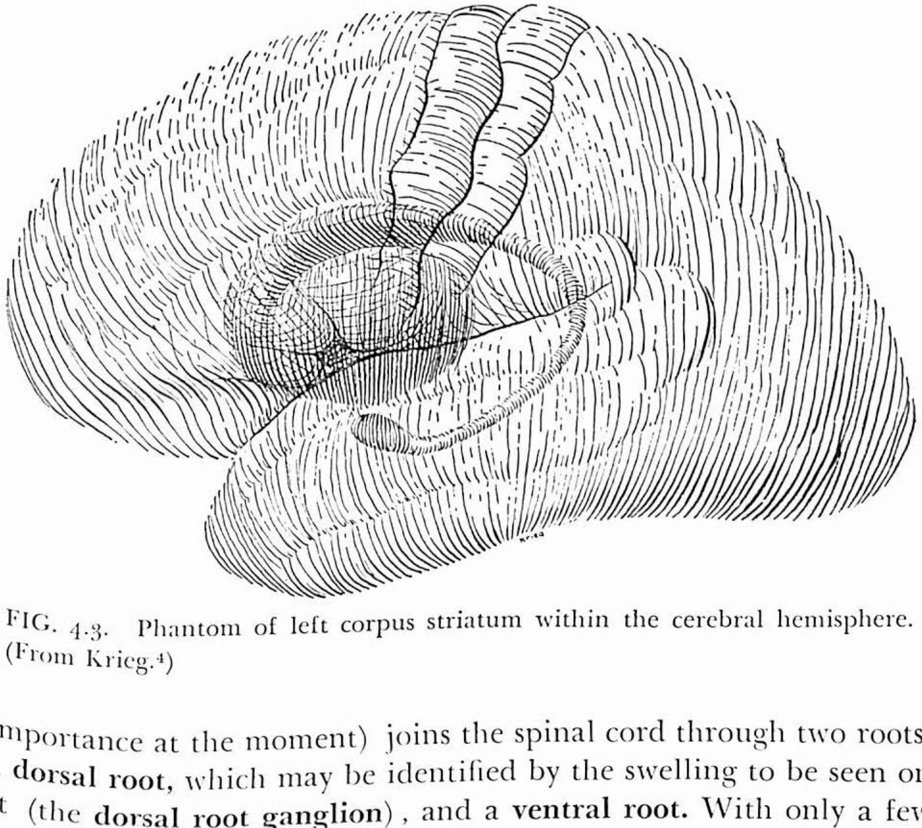


FIG. 4.3. Phantom of left corpus striatum within the cerebral hemisphere. (From Krieg.[4])

The two general functions of the spinal cord can be inferred from its structure. One is the conduction of impulses upward to higher centers and the conduction of impulses downward from higher centers. This conduction function permits the integration of CNS activities at all levels, and its interruption is, of course, very serious. The other function is a reflex one, associated with connections made within the cord (in the gray matter). We shall have much more to say about this function in Chapter 16.

The Brain Stem. The brain stem consists of several structures shown in Figure 4.2. If we proceed upward through the brain stem, we encounter first the **medulla.** Internally, the medulla looks very much like the spinal cord and its functions are similar — conduction and reflex coordination. Here we encounter for the first time the **cranial nerves.** A cranial nerve is one which arises from the brain itself. For easy reference, Table 4-1 shows the cranial nerves, their places of origin, and their most important functions.

Table 4-1. Summary of the Cranial Nerves

No.	*Name*	*Origin*	*Primary Functions*
I	Olfactory	Olfactory bulb	Afferent for smell
II	Optic	Diencephalon	Afferent for vision
III	Oculomotor	Midbrain	Afferent and efferent to all eye muscles except two
IV	Trochlear	Midbrain	Afferent and efferent to one eye muscle
V	Trigeminal	Pons	Afferent from skin and mucous membranes of head and from chewing muscles Efferent to chewing muscles
VI	Abducens	Pons	Afferent and efferent to one eye muscle
VII	Facial	Medulla	Afferent from taste buds of anterior $\frac{2}{3}$ of tongue Efferent to muscles of face and salivary glands
VIII	Acoustic	Medulla	Afferent from the inner ear (hearing and balance)
IX	Glossopharyngeal	Medulla	Afferent from throat, rear of tongue, and taste buds of posterior $\frac{1}{3}$ of tongue Efferent to throat and one salivary gland
X	Vagus	Medulla	Afferent from throat, viscera, and larynx Efferent to viscera
XI	Accessory	Medulla	Efferent to viscera (via Vagus) throat, larynx, and neck and shoulder muscles
XII	Hypoglossal	Medulla	Afferent and efferent to tongue muscles

Just above the medulla on the dorsal side of the brain stem will be seen the **cerebellum.** The cerebellum has an internal organization very different from that of the medulla and contains our first example of a true cortex, that is, the gray matter forms a rind or bark over the surface. The cerebellum has important functions in the coordination of movement (Chap. 18).

On the ventral side of the stem, opposite the cerebellum, the **pons** is found. Internally, the pons represents a continuation of the medulla, but on the surface the protrusion caused by the mass of fibers crossing from one side of the CNS to the other can be seen.

The next higher structure is called the **midbrain,** which is internally very similar to the medulla and pons. On the dorsal surface two pairs of small swellings may be seen. These are the **superior** and **inferior coliculi.** Often they are referred to as the **tectum** — in naming tracts leading to or from this region the adjectival form " tectal " is always used. The colliculi are important visual and auditory reflex centers.

The highest portion of the brain stem is the **diencephalon.** Ventrally this consists of the **hypothalamus,** which is an extremely important structure for visceral responses. Dorsally we find the **thalamus,** which is closely related to the **cerebral cortex.** Although the thalamus has other possible functions to be considered in Chapter 19, its importance as a sensory relay station must be mentioned here. With the exception of olfaction, all senses synapse in the thalamus on the way to the cerebral cortex. For each sense will be found special nuclei in the thalamus, and these will be noted when the senses are discussed in later chapters. The fibers from thalamus to cerebral cortex are called **projection fibers** and the areas to which they go in the cerebral cortex are called **projection areas.**

The Cerebrum. The **cerebrum** or **cerebral hemispheres** consists of two parts. Figure 4.3 is a phantom view showing one of these, the **corpus striatum** (or corpora striata, since there is one on each side). The corpora striata are very important in helping maintain posture and muscular balance. They will be encountered again in Chapter 18.

The other portion of the cerebral hemispheres is the cerebral cortex. This, our second and last, cortex is by far the more complex and important and an entire chapter (Chap. 19) is devoted to it. Suffice it to say here that it represents the highest level of coordination and control in the CNS, and to indicate the major subdivisions which will be mentioned in our discussions of the senses in later chapters. Figure 4.4 shows a lateral and a medial (midline) view of the cerebral hemispheres, with the names of the lobes shown. The surface of the cortex is folded, the ridges being called **gyri** (sing. *gyrus*), the grooves **sulci** (sing. *sulcus*). Two important landmarks on the lateral surface are the central sulcus, dividing the frontal and parietal lobes, and the lateral fissure, setting off the temporal lobe.

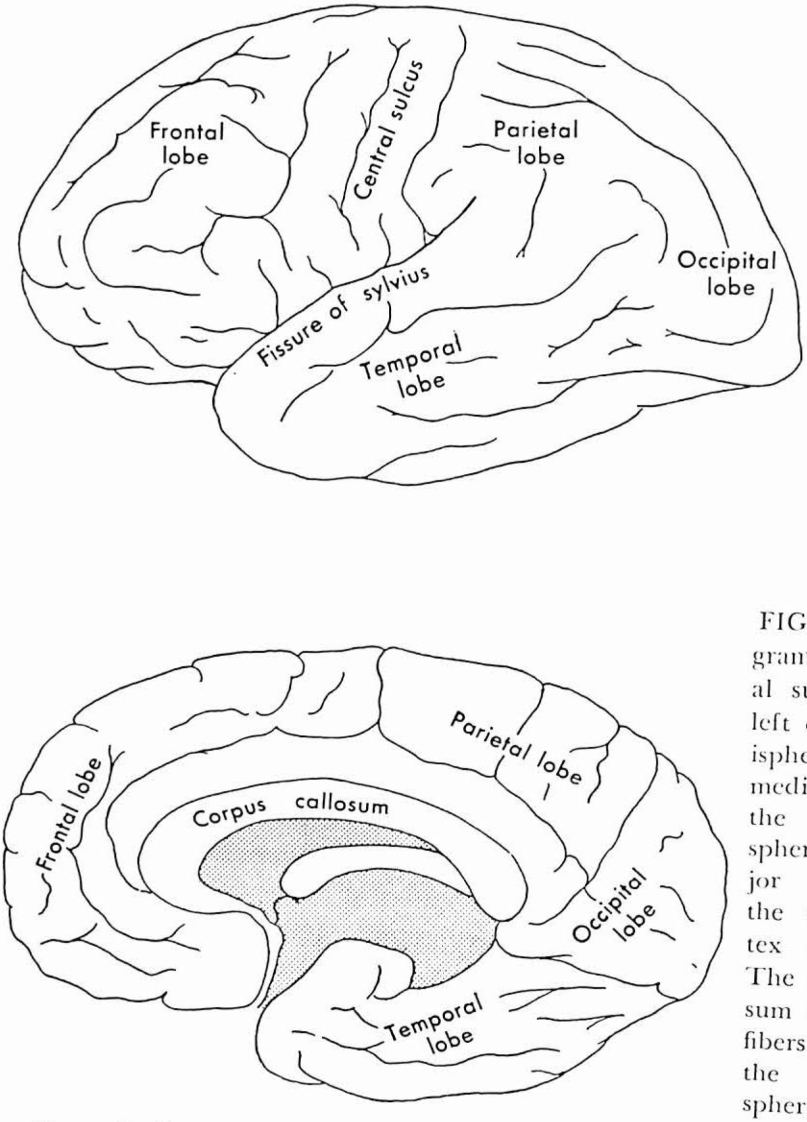


FIG. 4.4. A diagram of the lateral surface of the left cerebral hemisphere and the medial surface of the right hemisphere. The major divisions of the cerebral cortex are labeled. The corpus callosum is a mass of fibers connecting the two hemispheres.

Encephalization. The term encephalization refers to a principle so general that it requires introduction before we continue further. It refers to the fact that, during the course of evolution, the parts of the CNS most **rostral** are the ones which show the greatest development. Thus, the spinal cord has shown fewer changes from, say, shark to man, than has the brain; and only in mammals has the cerebral cortex gained pre-eminence. Related to this accelerated evolution of higher or more rostral structures is the development of their dominance over lower or more caudal structures. As an example, we may note that although the breathing centers of the medulla will function without stimulation from any other nervous structure, it is possible to hold one's breath for at least a time, which would not be

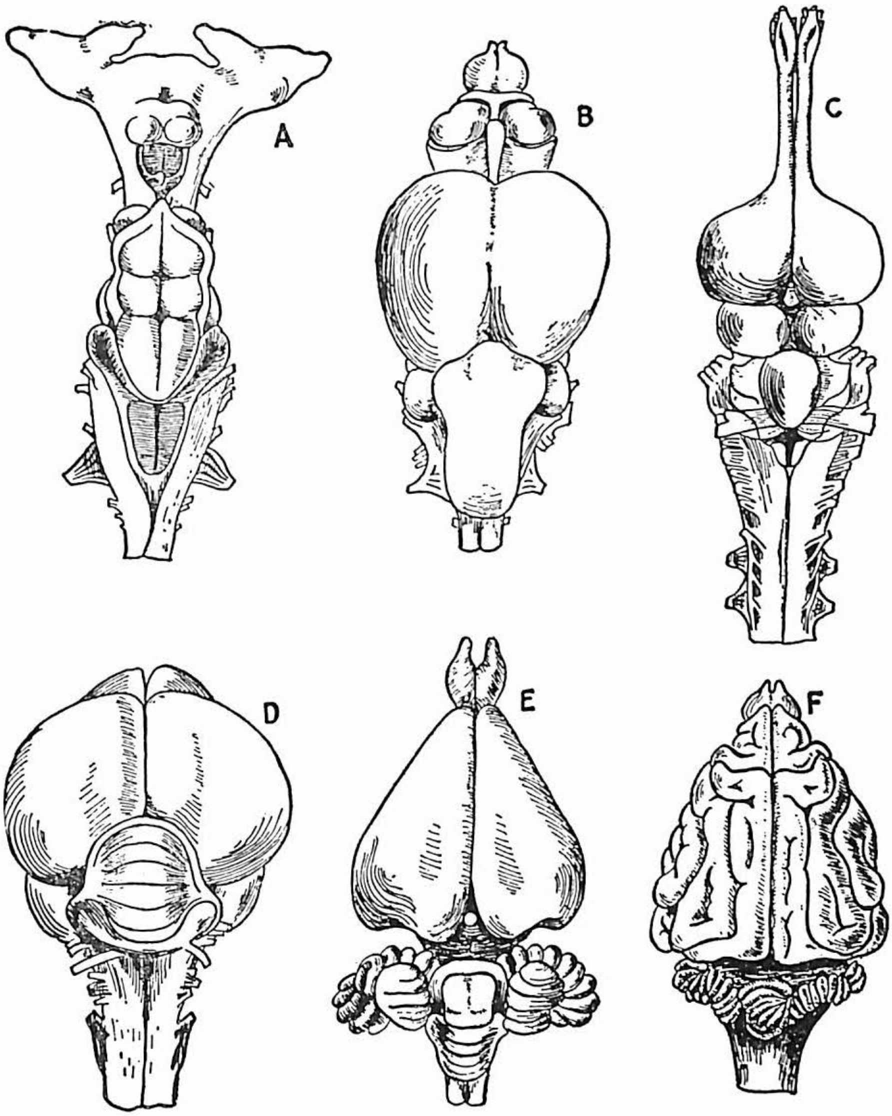


FIG. 4.5. Brains of vertebrates at different levels of evolution. Note the increase in the cerebral hemispheres. (From Mitchell.[5])

possible if the cerebral cortex did not exercise dominance over the medulla.

Interestingly enough, when the higher structures developed, the older ones did not disappear, and as a result the structures and connections to be found in so primitive an animal as the shark are still to be found in man. Encephalization involves the addition and dominance of higher structures, not the replacement of the older ones. The CNS is organized somewhat as is a system of bureaus, in which new bureaus have been added to supervise the functions of the old, but without the elimination of any. As a result, a given function of the CNS in man may involve several structures at different levels and often the only way of discovering the function of a lower, dominated center is by removing the higher structure and releasing the activities of the lower center from dominance.

EVIDENCE OF CNS FUNCTION

The student of psychology may wonder upon what evidence the statements concerning CNS functions, which are made in this and succeeding chapters, are based. It will not be possible to consider this point in detail, but we may consider briefly the principal methods of studying the CNS.

Ablation. The term ablation refers to removing or cutting out. When using this method, the experimenter removes some portion of the CNS, then observes the resulting loss or alteration of function. It is also used to release lower centers from the dominance of higher centers, as when the spinal reflexes are studied by cutting the spinal cord at its junction with the brain. When combined with accurate methods of observation of behavior both before and after operation, this method can yield very useful results.

Although ablation can be as gross or as limited as one wishes, much of the work done has been fairly gross. The experimenter may, for example, remove the entire cerebral cortex on one side, or remove some large structure such as the cerebellum. On the other hand, he may use delicate needle instruments to destroy small bits of tissue on the surface or deep within the brain. Also, observation of behavior can be casual and relatively uncontrolled, or can involve complicated measurements of particular acts. For example, after removal of the cerebellum we may note qualitatively that the animal is now unsteady and weak, or we may test him carefully both before and after operation and arrive at a quantitative statement of the change in behavior. This latter type of approach combines physiological and psychological techniques and has been a favorite method with physiological psychologists. Of course, the final step in the use of this method must be the very careful appraisal of the extent and nature of the tissue damaged, destroyed, or removed. This is accomplished by slicing and staining serial sections of the CNS and examining them under a microscope.

Strictly speaking, the method of ablation tells us what the parts of the CNS left behind can do, not what the part removed did. That a given function is only slightly or temporarily upset by the removal of a bit of tissue by no means proves that that bit of tissue was not involved in the given function. To give an example from a slightly different field, if a man can still see after losing one eye, we cannot assume that he did not use that eye when he still had it. When carefully interpreted, however, the method of ablation can yield very interesting and significant results.

Electrical Stimulation. Since the CNS is made up of neurons it can be stimulated electrically. This fact has been exploited very extensively in research. Not only can the exposed brains of animals be stimulated and responses noted, but also the brains of persons undergoing brain operations can be studied in this way. In the case of the human being, often only a local anesthetic is used and not only may we observe responses but, also, the patient can report on his sensations, perceptions, and so on.

One great advantage of this method, for animals other than man at least, is that the CNS being studied may be quite intact. On the other hand, when we stimulate electrically we cause all of the neurons in a given area to discharge simultaneously, which probably seldom happens in normal brain action. In other words, although we get positive evidence (as contrasted to the negative evidence derived from ablation) we do not get any information about the patterning of neural activity in the region stimulated.

Local Chemical Stimulation. When strychnine is applied to a nervous structure (e.g., the cerebral cortex), the neurons having cell bodies in the region of application will discharge repetitively. This selective action on cell bodies is an advantage which this method has over that of electrical stimulation (which affects both fibers and cell bodies). With strychnine stimulation we may be more certain that neurons located in the region being studied are involved in whatever functions are observed. It is more difficult, however, to confine the effects of strychnine and to control the strength of stimulation.

Typically, research with this method does not utilize behavioral responses but rather electrical recording methods (to be described below) in order to discover what other regions of the CNS are aroused by the strychninization of the region stimulated. Part of our knowledge of the interconnection of various cortical regions and of cortex and thalamus is based on experiments using this method. As a further extension, it is also possible to inject small amounts of strychnine into the deeper tissues by the use of hypodermic syringes. Sometimes strychninization is used to sensitize and perhaps synchronize the neurons in a given region, so that their response to activation through their normal fiber connections is made more readily recordable. Mapping the regions of the cortex aroused by sounds of different frequency was done in this way.

Electrical Recording Methods. Electrical recording methods may be used either alone or in combination with other methods and in either the laboratory or the clinic. Basically, this method is an extension of the methods used in recording the activity of a single neuron. When nervous tissue is active, it is possible to detect electrical potential changes, just as the passage of a nerve impulse is detected by the potential changes involved.

When a recording is made from outside the skull, the resulting record is referred to by the term **electroencephalogram** (translated literally: electrical-brain-record), abbreviated EEG. Obtaining an EEG is now a routine procedure in neurological practice. The relationships between the EEG and behavior will be discussed in later chapters. If we open the skull, it is possible to record from more limited areas, e.g., the surface of the cortex (electrocorticogram), or the thalamus (electrothalamogram). When recordings are made from outside the skull, only the synchronous activity of many neurons will give rise to potentials large enough to be detectable. Recordings from the surface of the brain, or from the deeper tissues, may, therefore, be considerably more precise than the EEG.

The Clinical Methods. Virtually all the work on the CNS of man has been done in connection with the treatment of disease or injury. Therein lies both the great advantage and the great disadvantage of this method. On the one hand, it does deal directly with man in whose CNS we are presumably most interested. On the other hand, it deals with disordered nervous systems. Unfortunately for the research neurologist, these disordered nervous systems represent neither clean-cut disorders, restricted to reasonably small areas, nor **lesions** in areas of interest for the research problem at hand.

In actuality, the clinical method refers not so much to a distinct method as to a place. In the clinic, all of the methods listed previously are used — ablation in some disorders, electrical stimulation, electrical recording, and even strychninization. Nevertheless, the basically nonexperimental character of the situation sets it apart.

Clinical evidence must be evaluated with the utmost caution. One seldom knows what the patient was really like before his disorder and no amount of subsequent study can remove this difficulty. Also, disorders seldom affect localized areas and in many cases (**tumor**, for example) pressure within the skull gives rise to widespread destruction, often far from the site of the original disorder. When, therefore, clinical and experimental results are contradictory, very careful examination of the exact circumstances of the clinical work is in order.

The Use of Lower Animals. Purely experimental methods are not used on normal persons, since the neurologist has an ethical obligation to seek the best interests of his patients. Obviously such experimental methods are applicable only to animals other than man and this involves some difficulty when we come to apply these experimental results to man. Needless to say, we can never be exactly sure how far our results will transfer directly. The more closely related to man the animal experimented upon, the smaller this hazard; therefore, chimpanzees and monkeys are more and more often used in experiments, although a great deal of the work is still done on cats and dogs. By now, many of the similarities and differences among the most-studied animal species have been analyzed, and so transfer of results from one species to another can be tempered by this knowledge. At any rate, there is no reason to suppose that neural activity is essentially different from one animal to another and the very much higher degree of control made possible by the use of animals more than insures the value of such research.

Suggested Readings

1. Kuntz, Albert, *A Textbook of Neuroanatomy,* 5th Ed. Philadelphia: Lea and Febriger, 1948.
2. Mettler, Frederick A., *Neuroanatomy,* 2nd Ed. St. Louis: C. V. Mosby Co., 1948.

5 | *Contracting Cells and Muscle Action*

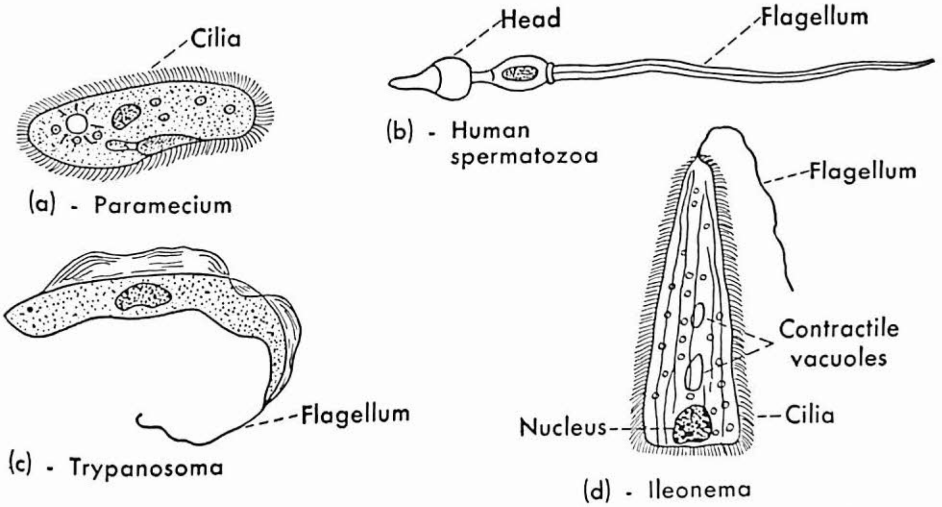
As students of psychology you are interested in behavior and, although behavior occasionally involves doing nothing, for the most part it consists of doing something. As far as organisms are concerned, behavior usually involves **muscle** activity — activity of groups of particular cells that have become differentiated for contraction.

These specialized contracting cells, along with the secreting cells which form our glands, are known as effectors. Glandular secretions are important, of course, and we shall consider them and their effects on behavior in a later chapter; but note the phrase, "their effects on behavior." In other words, the effector cells that secrete are of significance in physiological psychology mainly as they determine or alter the activity of effector cells that contract. These contracting cells, grouped in tissues known as muscle, are responsible for our every act and they even are responsible for some occasions when we refrain from acting. It is important, then, that we know something about them.

Primitive Contraction. The cells surrounding the pores of the sponges were among the first to become specialized for contraction in evolutionary history (Chap. 3). They illustrate the primitive muscle cell which responds directly to stimuli without an intermediate nervous system, but they do not represent the most primitive protoplasmic differentiation for contractility. This development is illustrated by the slender hair-like projections found on many single-celled organisms, which contain one or more tiny threads or fibrils that are the contracting elements.

Short projections of this sort are known as **cilia** and are found on many protozoa, where their fast beating action enables a much more rapid locomotion than that demonstrated by amoeba. Longer projections, or **flagella,** also occur on protozoa and both forms are found in many metazoa (see Fig. 5.1). Even in man, flagella provide for motility of sperm cells; and cilia line some of the passages of the body such as the respiratory tract, where their beating action assists in propelling dust or other particles away from the lungs.

Primitive Locomotion. We already have considered the most primitive form of locomotion when we discussed the movement of amoeba. This amoeboid movement, resulting from changes in tension in the cell membrane and related structures, is characteristic of many protozoa and is demonstrated by certain cells in our own bodies, such as some of our blood cells.



(a) - Paramecium

(b) - Human spermatozoa

(c) - Trypanosoma

(d) - Ileonema

FIG. 5.1. Examples of organisms depending upon cilia and flagella for locomotion.

In organisms with cilia or flagella, locomotion is brought about by a forceful contraction of the fibrils; and it is followed by relaxation of the fibrils and a subsequent return to their precontraction position, due largely to the elasticity of the sheath which surrounds them. Any smoothness in ciliary or flagellar movement is attributable to the fact that many fibrils are available and groups of them are contracting at different times — i.e., they are contracting intermittently, or asynchronously. Changes in direction of locomotion are due to the contraction of different groups of fibrils. But the fibrils that contract do so in an all-or-none manner and they exert their force in contraction, not in relaxation. Locomotion thus tends to be jerky and sporadic.

Even in the jellyfish, with nerve-net and with groups of specialized contractile cells, the apparent smoothness of swimming behavior is partially accounted for by the resistance of the water in which the activity occurs and only partially by the operation of opposed contractile forces. In the earthworm we find smooth, even locomotion;

it occurs chiefly by means of two sets of opposed muscles. One set is longitudinal in the body and the other is circular. When the longitudinal set contracts the body is shortened; when the circular muscles contract and the longitudinal muscles relax, the body is lengthened. The presence of a nervous system with a primitive brain is the important factor in the gradations of contraction that occur in these two groups of muscles, but the opposed muscles permit smooth coordinated locomotion when integrated by nervous control.

Another development of note in the earthworm is the rudimentary feet, which are short spiny projections pivoted in the body wall and which are moved by the contractions of muscles around their inner ends. Here they are not used to propel the organism but rather to hold a portion of it during body shortening or lengthening; and they are partially responsible for changes in direction of locomotion. They illustrate primitive forms of levers which are found in one form or another in most organisms with opposed groups of muscles. In higher organisms, practically all locomotion is mediated by pairs of muscles attached to skeletal structures that operate as levers, regardless of whether the form of locomotion is swimming, creeping, walking, running, or flying.

Three Kinds of Muscle. In all mammals three kinds of muscle tissue are found.

(a) The striped or **striated** muscles, which usually are attached to parts of the skeleton and which derive their name from the alternate light and dark striations which they demonstrate under the microscope. They contribute about one-half the weight of the body and are responsible for the movements of the body.

(b) The **smooth** or unstriated muscles, which for the most part are located in the viscera or the blood vessels and which appear to be more primitive in structure and function than striated muscles. They are responsible for most of the vegetative functions — those that are involved in the mere process of living, such as the activity of the stomach and bowels, the urinary bladder, and changes in size of the blood vessels.

(c) **Cardiac muscle,** which is similar in appearance to striped muscle and is found only in the heart.

Figure 5.2 shows these three kinds of muscle cells and the characteristics which give the first two their names.

Striped muscles are sometimes referred to with considerable accuracy as "skeletal muscles," but one has only to remember that such muscle is responsible for eye and tongue movements, as well as facial expressions, to realize that more than mere skeletal movement is involved. An even less accurate designation is " voluntary muscle," and the substitution of " involuntary muscle " for smooth muscle also is inappropriate. Although it is true that most striated muscles are more easily brought under voluntary control than most smooth muscles, the fact remains that few people are able to control the striped muscles that wiggle the ears, while some are able to exercise considerable control over the smooth musculature of the stomach and bowels (the Yogi for example), and most of us are able to control at will certain of the smooth muscles involved in urination. Furthermore, many reactions of striated muscles are completely involuntary, such as the maintenance of posture or the prompt withdrawal of a finger from a hot surface. All in all, it is better to think in terms of striated and smooth musculature, which will be seen to have quite different characteristics. We shall consider in later chapters the problem of learned muscular reactions and the significance of learned but involuntary control over both striated and unstriated muscles.

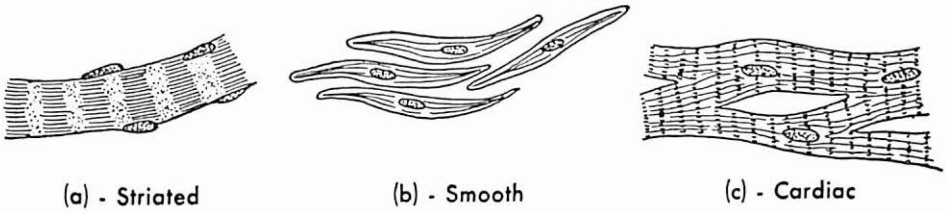

(a) - Striated (b) - Smooth (c) - Cardiac

FIG. 5.2. Types of muscle cells.

General Properties of Mammalian Striated Muscle Tissue. All mammalian muscle cells contain one or more thread-like fibrils similar to those found in cilia or flagella and here they are known as **myofibrils.** They are regarded as the contracting structures and they contain a special chemical substance, **actomyosin,** which rearranges its chemical structure upon stimulation and, thus, converts potential chemical energy into mechanical energy. Groups of muscle cells or fibers are surrounded by sheaths of connective tissue and constitute a **muscle bundle,** and a number of attached bundles make up a muscle.

Muscle cells demonstrate marked conducting as well as contracting properties and their conduction may be shown by appropriate electrical instruments just as it has been shown for nerve cells. The spike potentials obtained appear similar to those found in neurons; records of such action potentials in muscle, whether from a single cell or from a muscle group, are known as **electromyograms** (see Fig. 5.3). Both conduction and contraction occur upon direct stimulation by any of the energy changes which elicit conduction in a nerve cell, or they occur upon stimulation of the muscle fibers by nerve impulses of efferent, or motor, neurons. Moreover, muscle cells, like nerve cells, normally respond in an all-or-none fashion to a single stimulus.

Also, in common with nerve cells, muscle cells show several of the same metabolic phenomena; they utilize glycogen, they liberate heat and CO_2, and although they may respond in the absence of oxygen it is necessary for their recovery and subsequent response.

Properties of Smooth Muscle. Surprisingly, little is known of the operation of unstriated muscle in view of the fact that it is so important in our daily living. Each cell contains one nucleus and one or more myofibrils which are devoid of striations. The cells are combined in flat bundles or sheets and occur around the blood vessels and around most of the hollows or tubes of the viscera. Their unique property is maintained contraction which may be increased or decreased only slowly.

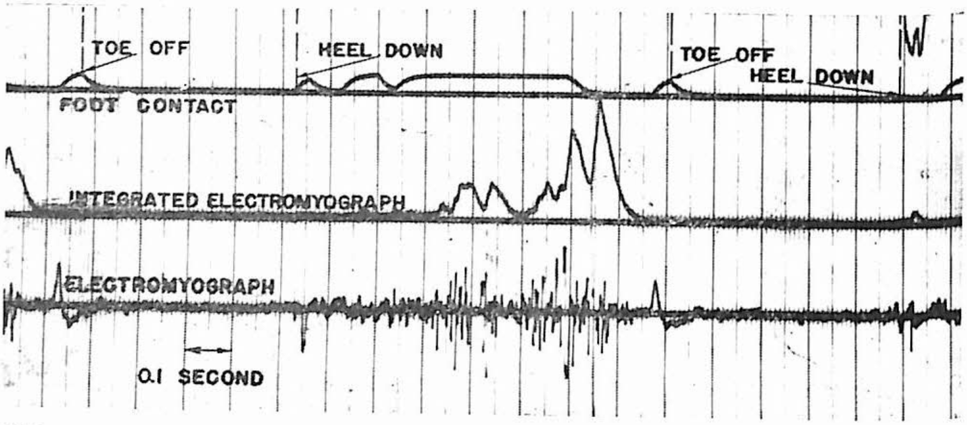


FIG. 5.3. Electromyographic record taken from a leg muscle group during walking. (From Fundamental Studies in Human Locomotion.[1])

In some instances, such as around the hair cells of the skin, the muscles are usually quiescent and contract only after appropriate stimulation; and in this case their response effects erection of the surface hair — **the pilomotor response** — and in animals with few surface hairs the contraction may be seen as "goose flesh." In other instances, such as in the stomach and the rest of the gastrointestinal tract, they engage in more or less continuous rhythmic contraction known as **peristalsis.** In other structures, such as the pupil of the eye, two completely separate smooth muscle tissues are in a state of continuous and opposed contraction. Additional stimulation of one pupillary tissue (the radial muscle) will dilate the pupil, while additional stimulation of the other (the circular muscle) will constrict the pupil.

In a later chapter, we shall consider many of the important functions of smooth muscles. For the present, it is sufficient to know that they are involved indirectly in much of our behavior and to know something of the manner in which they are innervated.

The Innervation of Smooth Muscles. Whenever a neuron is in such relationship to an effector that impulses in it will elicit reaction in the effector, the latter is said to be **innervated.** All smooth muscle tissues in mammals, and, also, heart muscle and the various secreting glands, are innervated by one or both of two groups of nerves which we shall come to know very well. Together they form the peripheral part of the **autonomic nervous system** (ANS), a system which is concerned with the regulation of basic bodily processes.

The ANS is not to be thought of as separate from CNS, since a major part of it lies within the spinal cord and brain stem and since the hypothalamus in the diencephalon is a major center for the integration of autonomic responses. The peripheral nerves of this system are known to liberate at least two chemical substances at their axon terminations: acetylcholine, which you already know, and **sympathin.** It is believed to be these chemical substances that effect a response in smooth muscles and it is known that chemicals similar to them will directly effect responses in certain smooth muscles and glands.

One of these similar chemical substances, **adrenalin,** is secreted by a gland innervated by the ANS. It can circulate in the blood stream for some time and affect many tissues. Thus, a widespread response may occur among all tissues that are reactive to adrenalin. Here is a partial explanation for the various smooth muscle responses that occur in a behavior pattern such as fear — the dilation of the pupil of the eye, the fast-beating heart, the pilomotor response, the sweating, and the constriction of peripheral blood vessels which produce pale and cold skin. This and many other patterns of behavior are determined largely by the effect of ANS stimulation upon smooth muscles. We shall consider this kind of behavior more thoroughly in the chapters dealing with autonomic functions and with emotional behavior.

Properties of Cardiac Muscle. Little need be said about cardiac or heart muscle in this text except that, though similar in appearance to striated muscle, its structure involves myofibrils that seem to branch and fuse with each other so that a network is formed throughout the muscle tissue. Contraction initiated in one portion of the heart is thus conducted to all other parts; and heart muscle appears to respond by waves of contraction. It is interesting to note that heart muscle possesses the capacity for restimulating its own rhythmic activity, that it is capable of continuous rhythmic response, and that it is the first effector to show action in the developing mammalian embryo. Even though cardiac muscle resembles striated muscle in appearance, it may be regarded, for the purposes of our study, as though it consisted of smooth muscle. It is innervated by the ANS and later we shall consider its innervation and response patterns in greater detail.

Properties of Striated Muscle. Much more is known about the characteristics of striped muscle tissue than about the other two forms. Investigators have been most interested in it because it is responsible for most of the observable behavior of higher organisms. Furthermore, most of it lies close to the skin and is easily accessible and almost any of it can be destroyed without marked interference with the basic metabolic functions of the organism. With the exception of some of the muscles involved in respiration, striated muscle tissue is not essential to life — it merely aids living.

Striated muscle tissue is composed of slender cells varying in diameter from 0.01 to 0.1 millimeters, and in length from a few millimeters to several centimeters. Lengthwise in the cells may be seen the myofibrils which have alternating light and dark portions that contribute the striped appearance to the muscle tissue. Each cell has several myofibrils and several nuclei and is surrounded by a membrane or sheath known as a **sarcolemma.** A number of cells joined and covered by connective tissue form a muscle bundle and these bundles are attached at either end to parts of the skeleton, to the skin, or to other muscle bundles, by connective tissues known as **tendons.**

Striated muscle cells are unique in the body in that they do external work — that is, mass is moved, whether in locomotion, in moving some body part, or in moving objects in the environment. They accomplish work by shortening their length under maintained tension, which is called **isotonic contraction.** As you lift a pencil, for example, your muscles are engaging in isotonic contraction. Your maintained hold on the pencil permits of little further shortening in finger muscle fibers, yet it is obvious that muscular activity is being continued if you do not drop the pencil. This kind of maintained contraction without further shortening is known as **isometric contraction;** it is the kind utilized in opposing gravity or other forces, and in maintaining upright posture. Both kinds of contraction generally are involved in manipulative behavior, i.e., in altering the environment, but only with muscle shortening does work occur.

If you will rest one of your arms on a table at a right angle from your body you will be able to demonstrate to yourself several characteristics of striated muscles during relaxation and contraction. First, relax your upper arm muscles and feel with your other hand their flabby consistency. If they are not soft and pliable, it means that you are not entirely comfortable, not entirely relaxed, under "strain." Only if the weight of the arm is completely supported by the table, and only if you are completely "at ease," will all residual isometric contraction disappear. Now tense your arm muscles without moving the arm. The firmer consistency of both biceps and triceps muscles (see Fig. 5.4) may be felt in their isometric, or constant length, contraction. Now lift your forearm, which occurs by means of isotonic contraction of the biceps, and rest your hand against your forehead. If you are in a comfortable position, your biceps will be relaxed somewhat, but probably not as much as when the arm was on the table. Some isometric contraction continues.

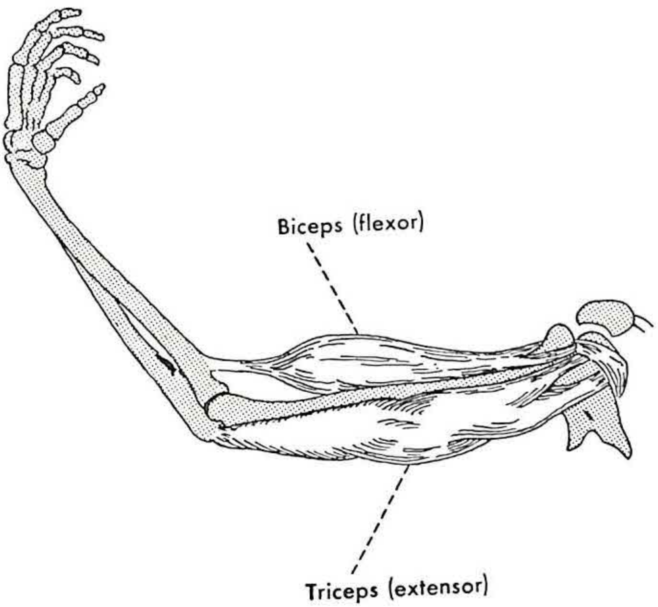


FIG. 5.4. Major opposed muscles of the upper arm in man.

Now, as you lower your arm, do it slowly and arrest its descent for a moment at about a 45° angle. It should be obvious to you that the process of lowering the arm has resulted in increased contraction in both triceps and biceps. The process of arresting movement involved a relative increase in biceps contraction until it exactly compensated triceps contraction. Had the biceps not contracted at all your forearm would have thumped the table with quite a blow. Had it been in maximum contraction you could not have lowered your arm.

Now look at Figure 5.4 again and note that the biceps is identified as a **flexor** muscle, because it flexes a joint or pulls a part of the body inward; and note that the triceps is called an **extensor,** because its contraction extends a part of the body. Notice, also, that the functions of this pair of muscles are opposed or antagonistic. All extremities of our bodies possess similar pairs of antagonistic striated muscles and it is only through a nice balance in their contractions that smooth coordinated movement is possible. This balance of contraction in antagonistic muscles is brought about by a balance in nerve impulses known as **reciprocal innervation.** We shall consider later how reciprocal innervation occurs.

Striated muscle tissue is unique in one other respect; it is specialized for rapid response, but it cannot maintain contraction over long periods. It is true, of course, that some striated muscles demonstrate rather continuous contraction in the maintenance of posture, but even these antigravity muscles cannot respond indefinitely and must be rested.

Innervation of Striated Muscles. Striated muscle tissue is innervated by peripheral efferent neurons which are myelinated and which are not a part of the ANS. Certain class labels have been used for them but none is entirely satisfactory and, in general, it will be best to refer to them merely as efferent fibers to striated muscle. When we come to a study of the ANS, we shall discover other means of distinguishing between fibers that innervate smooth and striped muscle. For the present, it is sufficient to know that all of the latter possess myelin sheaths and that they course directly to muscles from **motor muclei** in the **ventral horns** of the spinal cord or brain stem, where their cell bodies are found.

Certain similarities exist between neurons and striated muscle cells, and the effect of one neuron on another is similar to the effect of a neuron on a muscle fiber. Just as the synapse is a functional contact between neuron and neuron, the **neuromuscular** or **neuromyal junction** is a functional contact between neuron and striped muscle cells. It occurs between the axon terminal and a specialized receptive area of the sarcolemma known as the **motor end-plate** (see Fig. 5.5). As is the case with the synapse, the exact nature of conduction at the neuromyal junction is in doubt. There is no doubt that ACH is secreted at this point at the time when excitation takes place, but its exact function in excitation is unknown. A potential change also takes place at the end-plate and it is usually followed by an action potential of the muscle cell which elicits contraction of the fibrils. The normal course of events then is: nerve impulse — neuromyal transmission — end-plate potential — conduction in muscle — contraction in muscle.

The neuromyal junction does show functional properties which are not the same as those found in either nerve or muscle alone and, therefore, imparts characteristics to neuromuscular activity which are not derived from either nerve or muscle. It is usually considered that the junction is the locus of the fatigue which arises when a muscle cell is stimulated repetitively through its attached motor fiber, since transmission will fail at the junction while action potentials are still being recorded from the neuron and while the muscle cell is still directly excitable by an electric shock. Furthermore, the blocking action of drugs such as **curare** is confined to the junction and does not affect either muscle or nerve. Both fatigue and curare effects will be discussed in later chapters.

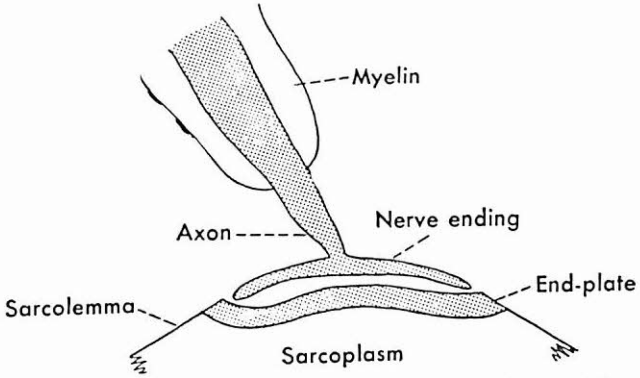


FIG. 5.5. Diagram of neuromyal junction. (After Couteaux.[2])

The Motor Unit. It is known that there are many more striated muscle fibers in the human body than the number of myelinated nerve fibers that innervate them. It is obvious, then, that one nerve fiber generally must innervate many muscle fibers. We find, however, great differences in the **innervation ratio** for different muscles. The ratio may be as small as 1:3 for some of the small muscles which mediate quick, precise movements such as those of the eyes; and for some of the large antigravity muscles involved in the maintenance of posture the ratio may be as great as 1:165 and is believed by some investigators to be larger. Each muscle cell appears to be served by only one axon branch. Thus, a neuron with a group of muscle fibers innervated by its several axon terminals may be thought of as a functional neuromuscular unit, or **motor unit.**

The number of motor units in striated muscles varies considerably and includes a range of at least 200 to 650. In view of the fact that both muscle and nerve cells respond in an all-or-nothing fashion, it should be obvious that the number of motor units in a given muscle and their innervation ratios will influence the precision and range of contraction of which the muscle is capable. Muscles with small innervation ratios and many motor units are capable of fine control over a wide range of contraction. Muscles with large innervation ratios and few motor units are less capable of precise or graduated action.

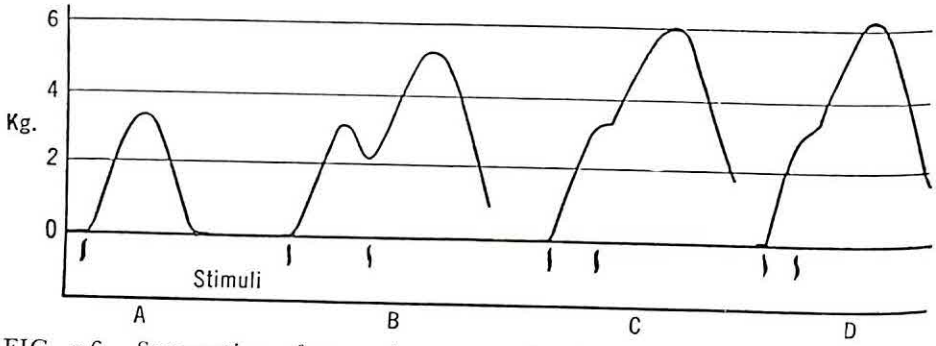


FIG. 5.6. Summation of muscular contraction by successive stimulation. Interval between stimuli are B = 57 m sec.; C = 40 m sec.; D = 24 m sec. (After S. Cooper and J. C. Eccles.[3])

Summation and Asynchronous Innervation. Even in muscles with many motor units of low innervation ratio, the response would be jerky if all units were to discharge synchronously at a slow rate. If a motor unit is activated by a single stimulus, the result will be a single contraction known as a twitch, which then is followed by relaxation. If a series of separate stimuli are applied at a rate slower than about 5 per second, the result will be a series of twitches known as **clonus,** which involves alternate and jerky contraction and relaxation. Clonic activity and single twitches may occur in the intact human organism, but ordinarily they are the result of some disorder. The typical pattern of even contraction of muscle groups, known as **tetanus,** is the result of rapid asynchronous innervations which are summated.

Summation in muscle contraction may best be understood by consulting the diagrams in Figure 5.6. In part A, a single muscle twitch is shown; in B, a second stimulus has been applied before relaxation following the first stimulus was complete, and a second twitch is superimposed on the first; but note that the magnitude or strength of the second twitch is greater than that of the first. This observation does not serve to deny the all-or-none principle. It is similar to stimulation of a neuron during the period of superexcitability; the conditions of stimulation are different and the response, though maximal in each case, is different. At C of the diagram, the interval between stimuli has been decreased further and the two twitches are practically fused; while at D, fusion is complete and the tetanus is seen to be much stronger than the single twitch.

Summated tetanic contraction is the typical response of striated muscle in intact organisms, for even a single stimulus applied to a receptor cell will set up a series of afferent impulses (Chap. 6). These impulses, occurring at or near the maximum rate at which nerve fibers can conduct, are well within the time requirements for tetanic muscular contraction. Furthermore, the impulses in different nerve fibers are asynchronous. Even though many are begun simultaneously at a receptor organ, the rates of conduction will vary considerably throughout the nervous system, so that asynchrony would be expected by the time the impulses affect motor units, which themselves vary in discharge rate.

Even muscular response is brought about, then, through many characteristics of neuromuscular structure and function. The major factors are (1) the innervation ratio, (2) the number of motor units involved, (3) the summation which makes possible the maintained contraction of tetanus, (4) the asynchronous innervation and discharge of motor units, and (5) the reciprocal innervation of opposed muscle groups.

Muscle Tension. So far in this chapter we have not used the familiar term "muscle tension." You have seen that muscle cells respond by contracting and that they have two main kinds of response — the twitch and tetanus. Subtetanic contractions are possible and when they occur a series of fractional twitches or tremors can be detected; but in intact healthy organisms the typical response is tetanic contraction of one or more motor units. If this is a shortening or isotonic contraction a movement of some body part will occur, which is known as **phasic contraction.** If contraction is isometric, the tetanus is maintained and is known as **tonic contraction.** Although tension is exerted whenever a muscle contracts, the term "muscle tension" usually refers to tonic activity in muscle cells.

Both smooth and striated muscle tissues exhibit more or less continuous low-grade tension, known as tone or **tonus.** For smooth muscles this is due to more or less continuous innervation by the ANS; while for striated muscles it is due largely to reflexes brought about by the stretch placed upon the muscle bundles by their attachments. Health, exercise, and related metabolic factors also are involved in with severe sunburn, acquired on cool, hazy days. Another familiar example is that of radio waves; we are not sensitive to these as a radio is and we can only hear sound after the radio has transformed the signal into audible frequencies.

But what about our knowledge of ourselves? Is it any more direct or immediate than our knowledge of external objects, or do we likewise need special sensitivities in order to gain information about how things are going in the gut or how the legs are holding up on a strenuous hike? Let us take another example; that of a man who, through disease, has lost all sensation from the muscles of his legs, although the movement of his legs has not been impaired. Such a man can walk, but unless he looks at his legs, he doesn't know where they are or what they are doing, and in the dark he is almost helpless. A few unfortunate individuals who have lost sensations from their abdominal organs complain bitterly of being hollow and having lost their insides. Evidently we require sense organs to give us information about ourselves. We may largely ignore such sensations, unless intense because of some malfunction, but they exist as a constant backdrop, against which the play of external life goes on. They are difficult to verbalize and it requires practice to observe them properly, but they are one of the important bases for the perception of the self and of the feeling of well-being or illness.

The study of sensation has assumed great practical importance in recent years because it is basic to the control of machinery; moreover, our military forces, as well as our industries and private lives, are daily becoming more mechanized. In order to control any sort of machine, one must be aware of what is happening and this is dependent upon the **acuity** and other characteristics of one's sensory systems. Aircraft dials must be designed so that they can be read with a minimum of time and error. Should one use a clock dial or a horizontal dial? What color should it be? What intervals should be used? And many more questions of the same sort. If, as in some dials, it is easy to make mistakes of 1000 feet, the result may sometimes be fatal. Again, radar screens are very hard to read. What can be done to improve them? Or take the telephone; the clarity and distinctness with which you can now hear voices is partly a direct result of intensive study of the characteristics of human auditory sensory mechanisms. One of the reasons for the increasing number of automobile accidents is the overloading of the eyes in driving — the instruments to watch, the number of cars and pedestrians to watch. Many people do not have the necessary sensory skills for safe driving — their perceptual speed is slow, too slow for modern traffic conditions.

Sensation and Perception. For many purposes it is convenient to make a distinction between **sensation** and **perception,** although this distinction must not be considered a hard-and-fast one. Traditionally, sensation has been used to refer to those problems in which the functions of the bare sensory system itself were investigated, as opposed to more obviously perceptual problems of complex patterns and interrelationships. In some psychological theories, sensation has also been a construct, an element of consciousness derived from the mere functioning of the afferent system, to which other elements are added to make a perception. In actual practice, one never experiences a sensation, but only relatively simple perceptions. We discovered long ago that even in such situations as observing a homogeneous colored light, previous experience and the instructions to the subject make a difference. There is a continuum of perceptions, from simple to complex, but it is still convenient to separate the simple from the complex problems because of differences in experimental techniques. We will call the field of simpler problems the topic of sensation and the field of more complex ones the topic of perception.

It may be helpful in establishing this definition to give a few examples of representative problems in each field. Typical research in sensation is the determination of thresholds for color vision at each wavelength, of whether salt will partially cancel sour in taste, or of whether the sense of pain shows adaptation. Typical research in perception involves studies of color constancy, of the relative importance of factors of depth perception, or of the audibility of speech with various amounts of masking noise. In this book sensory problems will be stressed more heavily than perceptual ones for three reasons: (1) the simpler, sensory problems are basic and must be understood first; (2) they are usually treated very superficially in the beginning course; (3) they are more readily related to physiological psychology in our present state of knowledge.

Functions of Receptors. Receptors, or sense organs, are the first elements in the afferent pathway. They are of three general types as shown in Figure 6.1: (1) the afferent neuron may be unspecialized, merely branching freely at its endings, as in the pain receptor; (2) the afferent neuron may itself become highly specialized, as in the olfactory receptor; (3) the afferent neuron may be associated with specialized cells which assist it in performing its functions, as in the taste receptor. Although it is possible to stimulate an afferent nerve directly in an experiment, in the normal course of life it is a receptor which is stimulated and the receptor is then responsible for setting off the nerve impulse. Why is it that a receptor, simple or complex, is always inserted into the afferent system? What peculiar characteristics do receptors as a class have which renders them so important? The answers to these questions are not simple. There are three important characteristics of receptors. First, receptors are more sensitive than nerve fibers. This *extra sensitivity* means lower thresholds and, thus, ability to respond to lower levels of energy change in the environment. It is responsible for our ability to perceive slight differences and, thus, adjust to the highly complex patterns which are the basis of human life. Thus, it also acts as an important safety factor. Second, receptors are selective in the stimuli to which they respond. This *selectivity* means that each kind of receptor is specialized so that it is especially sensitive to one kind of energy change in the environment. The receptors in the eye have very low thresholds for light and have, in addition, accessory structures which bring light to them more readily — but they are not more sensitive than nerve fibers to sound waves. They are specialized for response to light and to nothing else. This selectivity means that greater differentiation is possible and adds immeasurably to our ability to perceive complex patterns. Third, receptors are responsible for *prolonged response* to a constant stimulus. A nerve fiber responds but once, or at most a few times, to a stimulus applied to it. But the receptor is capable of responding many times and, therefore, gives us information of a very important kind — that the thing is still there, or that it is becoming more intense. Imagine, if you can, a world in which each new object gave only one or two brief signals of its presence and then was " silent " — we would be virtually without the dimension of duration in our lives.

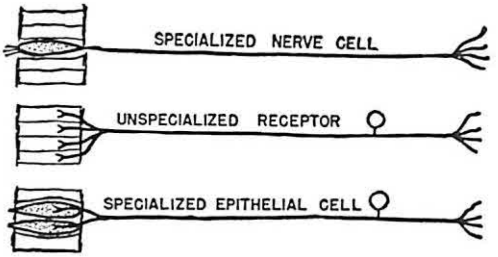


FIG. 6.1. Diagrams of three types of receptors: (A) specialized neuron. (B) unspecialized nerve cell, (C) specialized epithelial cell. (After Morgan and Stellar.[1])

Senses are often classified into groups and there are several ways of doing it, depending upon one's interest or purpose. There are four criteria which are generally used. First, a *psychological criterion* may be applied: do the sensations seem similar? Pressure and pain feel different, so on the basis of a psychological criterion we would classify them as separate senses. Second, we may use a *physical criterion:* are the receptors stimulated adequately by the same forms of energy? The eye and the ear respond to different forms of physical energy and would therefore be judged to belong to different senses. Third, an *anatomical criterion* may be used: are the sensory systems similar anatomically? Taste and smell are classified separately because the receptors, their origins, and their cortical terminations are different for each system. Fourth, a *physiological criterion* may be brought to bear: what kind of response is evoked? Is the response a postural one, a visceral one, or action upon the environment? Actually, all these criteria are used and there really is no hard-and-fast definition of a sense department. How different anatomically must they be before they are considered to belong to different senses, rather than to the same one? Each criterion emphasizes some different aspect of the topic and each is thus useful. By using all of them judiciously we will find that we have many more than the traditional five senses, but whether we enumerate nine, thirteen, or some other number depends upon the fineness of distinction which we choose to make.

A general classification of receptors which is often made is a division into four classes on the basis of all considerations. Table 6-1 shows the division and the senses normally falling in each. The divisions are not always sharp, as is indicated in the table. Gustation, although primarily an interoceptor, can also give information about external objects and was an important source of information for Helen Keller. Olfaction, although an exteroceptor in giving cues to various objects about one, also operates as an interoceptor along with taste. Pain is in a still more anomalous position, being in a class by itself (nociception) because of certain response characteristics, but also serving as both exteroceptor and interoceptor. The particular class used in any given instance depends upon the circumstances and one's purpose.

Stimulus. We have already used the term **stimulus** many times but this is the place to define it more carefully. The term is often used in two senses. The first is a rather general one which we have used earlier in the book — any agent that directly influences the activity of protoplasm. In the next section, dealing with sensation, the word is used in a narrower meaning — energy which excites a receptor. Thus, a change in some hormone level, although a stimulus in the general sense, is not a stimulus in the sensory sense as it does not excite a receptor. **Sensory stimuli** are such things as light waves, sound waves, objects at temperatures above that of the skin, muscle tension, and the like.

The concept of the **adequate** stimulus is one which you will meet

Table 6-1. General Classification of Receptors

Class	Sense	Basis for Classification
Exteroceptors	Cutaneous pain Cutaneous pressure Cutaneous warmth Cutaneous cold Vision Audition Olfaction Gustation	Receptors located primarily on body surfaces; respond largely to external stimulation; response is primarily bodily movement.
Interoceptors	Organic pain Organic pressure Organic warmth Organic cold Olfaction Gustation	Receptors located primarily within the body (digestive and urogenital tracts); respond largely to stimulation within the body; response is primarily internal change.
Proprioceptors	Muscle kinesthesis Tendon kinesthesis Joint kinesthesis Vestibular senses	Receptors located in muscles, tendons, and joints, and nonauditory inner ear; respond largely to bodily movement; response is primarily bodily movement.
Nociceptors	Cutaneous pain Organic pain Kinesthetic pain	Receptors located throughout the organism; respond largely to injurious stimuli; response is primarily flexion.

time and again in any discussion of sensation. An adequate stimulus is one which is normal for the sense organ in question. An inadequate stimulus is merely an abnormal one; it is *not* a stimulus which does *not* excite — it would not be a stimulus at all under those circumstances. Note that the dictionary definition of adequate does not apply here, because this is a technical term. Thus, the adequate stimulus for vision is light, but vision may be stimulated inadequately (abnormally) by electric current or a blow on the eye (seeing stars). The adequate stimulus for the cold receptors is a lowered skin temperature, but they may be stimulated inadequately by pressure, electric current, or a pin prick, if these are strong enough.

Most experiments in sensation, unless they are purely exploratory, require specification of the stimulus; that is, they require that the stimuli used be specified as exactly as possible. To be specified, a stimulus must be controlled, so that specification implies both control and quantification. In order to control, quantify, and, therefore, specify the stimulus, one needs information about the important variables of the particular stimulus — the dimensions along which that particular kind of energy can vary. These have been adequately described, for all practical purposes, by the physical scientists and we need only learn from them what to control and how to do it. In general, the important dimensions are amount of energy, time characteristics (frequency, and/or duration, wave shape, etc.), sometimes extent, and possibly pattern (in complex stimuli) for each kind of energy.

It is important that a sharp distinction be made between stimulus variables and sensation variables. In **psychophysics** we attempt to find a relation of some sort between the two; we cannot assume it to be perfect. We may want to know, for example, how the brightness of a light is related to the energy in the stimulus. In order to write this equation it is necessary to measure the stimulus in terms of its physical variables and the sensation in terms of its psychological variables. A sensation can never be described in physical terms; such a statement is meaningless. The physical dimensions are ways of specifying the stimulus, and this is the first step in the investigation of sensation, but only the first step. We shall discuss later in this chapter the psychological variables and their relationship to the physical dimensions.

Sensory Thresholds. The concept of the threshold is one which is derived statistically. In all scientific measurement there are small uncontrollable variations, so that no single measurement is truly representative and many measurements must be made. With a large number of measurements, which always vary somewhat, you need a statistical technique to describe the essential meaning of what you have found. Thus, a threshold, dependent upon measurement of many instances, may be defined as that stimulus which can be distinguished from some standard exactly 50 percent of the time. This definition is general and, therefore, harder to understand than the more specific definitions of the various kinds of thresholds. Let us turn to them for clarification.

There are three kinds of thresholds which sometimes are called limens: (1) the **absolute threshold** (RL), (2) the **difference threshold** (DL), and (3) the **terminal threshold** (TL).

The absolute threshold is the one lying at the lower end of any scale of intensities for a particular kind of stimulus. It is that stimulus which can be sensed or felt 50 percent of the times it is applied. In this case the " standard " is zero stimulation. The absolute threshold for pressure is that weight which is judged to be present, or is sensed 50 percent of the times it is applied — the other 50 percent of the times the subject does not feel anything.

The terminal threshold lies at the other end of the stimulus scale. It represents the level above which an increase in the stimulus energy yields no further increase in intensity 50 percent of the time. In the case of the terminal threshold, one may have a more complex judgment to make because it is often mixed with pain and sometimes it cannot really be measured. The terminal threshold represents statistically the effective upper limit we can sense, just as the absolute threshold represents the effective lower limit of our sensitivity. Absolute thresholds are always of immediate significance; terminal thresholds are so more rarely. The terminal threshold of greatest practical importance is that for audition, where the upper limit of intensity is taken as the point at which pain is felt — 50 percent of the time — and this is the upper intensive limit of useful sound.

The difference threshold is the difference between a given stimulus and another stimulus which is judged as different from it 50 percent of the time (sometimes, for technical reasons, a somewhat different cut-off point is used). The difference thresholds are those thresholds lying between the absolute and terminal thresholds. An example of a difference-threshold experiment is the determination of the amount of sugar which must be added to a given solution in order to make it just noticeably sweeter. If one begins at some particular point, such as the absolute threshold, one finds that the perception of differences of this sort is not continuous. It is not smooth, as the slope of a hill, but discrete, like an irregular staircase, upon which you have to rise a certain amount at each step in order to succeed in going from bottom to top. If the risers are 6 inches, you must lift your foot 6 inches in order to achieve the next step — it does no good to lift it 4 inches. Similarly, in a psychological scale, if the increase in, say, sweetness is to be perceptible at all, it must be one DL, i.e., it must equal the difference threshold. But let us not push our staircase analogy too far. We do not mean that in the DL "staircase" all the risers are identical. In this "staircase," the height of the risers changes — the absolute amount which must be added to a stimulus in order to produce a DL is not constant; it changes, depending upon the position in the scale at which one is working. One of the earliest concerns of the first physiological psychologists was with this problem of the relationship between the increase in stimulus energy and perceptible differences. In general, the absolute amount which must be added to a stimulus in order to produce a DL is larger, the more intense the stimulus which is taken as standard. The amount which must be added each time increases as we ascend the scale from absolute to terminal threshold; furthermore, it increases approximately in direct ratio to the standard stimulus. **Weber's Law** states that the amount by which a stimulus must be increased in order to be just noticeably different bears a constant ratio to the standard stimulus. The Weber fraction, which puts this more succinctly, is:

$$\frac{\Delta I}{I} = C$$

This states that the increment of intensity (ΔI) divided by the standard intensity (I) is constant. The Weber fraction is, of course, different for the various senses. For taste $\frac{\Delta I}{I}$ is about .2, for cutaneous pressure about .15. The general correctness of this relationship is obvious from casual observation. If you enter a room lit by only one lamp and turn on one more lamp, the room becomes very much brighter than it had been; but if you add one lamp to four already lit, the difference is scarcely noticeable. The one-to-one ratio is well above the difference threshold, the one-to-four ratio is not. Again, if you begin to play a piano in a quiet living room, the increase in sound is great, but the addition of the same piano to a full symphony orchestra would probably not be noticed.

Once the essential correctness of this relationship has been grasped, it is necessary to qualify it. The Weber fraction is not quite constant; it remains fairly constant in the middle range of intensities, but tends to increase more and more as it approaches the extremes. Figure 10.3 shows an actual plot of a Weber fraction from experimental data — the solid line. If the Weber fraction were completely constant the line would appear horizontal, as in the dotted line. Discussion of the ramifications of the Weber fraction would take us far afield, into some of the most sophisticated aspects of modern psychology. The most important point to remember is that the size of the DL varies and is usually larger for more intense stimuli than for weak ones.

Although thresholds are normally thought of in connection with intensity, there are also qualitative thresholds in some senses. In audition there are absolute, terminal, and difference thresholds for pitch; in vision there are difference thresholds for hue. Many of the problems are the same, but with qualitative difference thresholds Weber's law does not apply — it belongs to intensity alone.

Of what significance are thresholds? They are of both theoretical and practical importance. The measurement of a threshold testifies to the variability of the theoretical threshold. There are a number of factors involved in this variability, among the most important of which are: (1) errors of measurement, (2) periodic fluctuation in the sensitivity of the receptors and, probably also, other parts of the sensory mechanism, and (3) fluctuations in the attention of the subject, that is to say, in the central nervous mechanism. Threshold data are useful in many situations, some of which we have already indicated, others of which will be pointed out in connection with the discussion of the individual senses. In any situation, military, industrial, or esthetic, in which the absolute or differential sensitivity of people at large is important, the measurement of thresholds provides essential information.

Dimensions of Sensation. In the discussion of the stimulus, the distinction between stimulus variables and sensory variables was emphasized and the nature of the stimulus variables described. We are now ready to describe the dimensions of sensation, often called the *attributes of sensation.* These are the ways in which any given sensation may vary independently and they are four in number.

Quality. The first and most obvious dimension is *quality.* This is the unique characteristic which differentiates psychologically among different senses, as sound from color or warmth; and among different aspects of a single sense, as salt from sweet, or red from blue. It cannot be defined verbally; it can only be pointed to.

Intensity. The second dimension is one with which we already have a speaking acquaintance, *intensity,* and represents the "moreness" or "lessness" of a particular quality — a faint sound or a loud sound of the same pitch, a light red or a dark red of the same hue, a slight warmth or a great warmth. Intensity may thus vary independently of quality.

Extent. The third dimension is called *extent,* and refers to the "spread-outness" of some sensations — how large an area of pressure, how large an expanse of blue.

Duration. The fourth dimension is the time dimension, and we call it *duration.* It refers to how long a sensation lasts. Is it brief or prolonged?

All four of these aspects can vary independently, that is without influence upon the others, except, as you would expect, at the extremes. It is obviously possible to make an object so small that you cannot see it at all, and thus change quality by changing extent, but you do not change color by simply presenting a 2-inch square piece of colored paper instead of a 1-inch square piece. It is equally obvious that duration will have some influence upon the other dimensions at the extremes, as we shall see later. But it is still generally true that the four dimensions can be varied independently. These four aspects are ordinarily sufficient to describe psychologically any sensation, leaving out the perceptual aspects of meaning and feeling. Thus, if you are looking at an orange, what we choose to call the sensation can be described as a certain extent of yellow-red of medium intensity which endures quite some time, if you choose to look at it. Whether you know what it is, where it comes from, what people normally do with it, and whether you like it — these things are irrelevant to a purely sensory description of it.

Physiological Correlates. It is of some interest to inquire into the possible physiological correlates for these psychological dimensions. Quality does not reside in the object but in the organism. No object is ever red; but an individual looking at an object which reflects the long-wave end of the visible spectrum, if the light falling on it contains these waves, and if the individual has normal color vision (and several more ifs), will see red. You already know that the quality resides in the organism (see p. 68), for an inadequate stimulus is an abnormal one for a particular sense — pressure on the eyeball causes one to see light, for example. There are several lines of evidence which lead to the conclusion that quality is dependent upon the place in the cerebral cortex which is excited by afferent impulses. If the afferent impulses do not reach the cortex, no sensation is felt at all, as when the spinal cord is severed and reflexes are present but there are no sensations from the legs. If the cortex is stimulated electrically, certain sensations result from stimulation of certain regions. If tumors occur in particular regions, disturbances of a particular kind of sensations result. Special areas of the cortex are, then, correlated with qualities of sensation — occipital lobe with vision, temporal lobe with audition, and so on. We have not yet solved the more difficult problem of qualitative differences within a single sense department, but it is asumed to be a still finer spatial differentiation, red and blue perhaps occurring at different depths within the cortical layers of the occipital lobe.

The physiological correlate of intensity has been worked out rather carefully. It is evident from the discussion of the all-or-nothing law of nerve action in Chapter 3, that intensity cannot be represented by larger nerve impulses. What happens, then, if a more intense stimulus is applied? Two things: (1) the nerve fiber is excited earlier in the relative refractory period, and (2) more receptors (those with higher thresholds) are brought into play. The net result of these two events is that more impulses arrive at their cortical station per unit time. Intensity may thus be said to be related to the number of impulses per unit time (e.g., per second) arriving at the cortex.

Extent and duration are both more obvious and less certain. Extent appears to be correlated with the physical extent of cortex excited, a mere size for size translation. And duration appears to be correlated with duration of excitation in the proper area of the cortex. But there is little actual evidence and these assumptions are undoubtedly too naïve to be strictly accurate. The interrelationships among all four of these dimensions and their underlying physiological correlates are exceedingly complex and it must be realized that the above account is highly simplified.

Physical Correlates. The physical correlates of sensation are better known, although not as simple as one might expect. Psychophysics is the attempt to state these relationships between the stimulus (physical variables) and the sensory variables. It may be taken as axiomatic that there is no one-to-one correlation between the two, either for quality or intensity. For quality, the physical correlate is the kind of stimulus. Electromagnetic radiation of certain frequencies, commonly called light waves, gives us visual qualities, vibrations of certain frequencies, commonly called sound waves, give us auditory qualities, and so on. Within the visual system itself, quality (redness, blueness, etc.) is generally correlated with wavelength, but this is true only within certain limits of energy. Intensity, as a psychological dimension, is correlated with energy in the stimulus, but other things are important also. If an object is vibrating at a frequency beyond our audible range, say 30,000 cycles per second, no energy, no matter how great, will give it any intensity — it remains inaudible. The physical correlates of extent and duration are obvious, for we have borrowed the terms from them. Even here, however, extent changes somewhat with energy, bright objects appearing larger than dim ones of the same size, and very brief flashes of light appearing dimmer than longer ones. Thus, we see once more that the interrelationships are complex.

Characteristics of the Organism. It probably seems like a truism to state, at this point, that it is the characteristics of the organism which are ultimately the determiners of sensation. This chapter began with a discussion of the importance of the limits of man's sensitivities to his ability to respond to his environment. This point must now be expanded. In every sense department there are limits to man's sensitivity. In vision, we cannot see radiation of very short wavelength, the ultraviolet waves, nor can we see the infrared waves. There is a continuity of radiation from one through the other; the limits lie in the organism, not in the physical world. Similarly, there are vibrations too rapid for our ears — they lie outside our limits of sensitivity. And there are temperatures too low to affect our cold receptors. The sensitivity of the organism determines what we shall sense.

Another characteristic of the organism which determines sensation is the state of *adaptation*. With continued exposure to certain kinds of stimuli the organism is so changed, by adaptation, that he no longer senses what he did previously. The first plunge into a lake excites the cold receptors, but after a few moments they no longer respond. If one stays in a room illuminated by yellow, incandescent light and then observes a piece of red paper under neutral light, it will appear purplish. The organism is changed by adaptation, and this state of adaptation determines in part what one will sense. A third factor which will affect sensation is the presence of surrounding or accompanying stimuli. A gray square surrounded by a large area of red will appear greenish, but the same gray square surrounded by blue will appear yellowish. Subliminal sweet on one side of the tongue is made perceptible by application of a sour stimulus to the other side of the tongue. Thus, the characteristics of the organism determine what is sensed. No stone is really warm, no apple really red, no drink really bitter. The stone is at a particular temperature and whether or not it will feel warm depends upon the characteristics of the organism that touches it — if the temperature mechanisms have been destroyed by disease, or the hand adapted to that temperature, it will not feel warm. If the man observing the apple is color-blind, or has been staring at a yellow wall, the apple will not be red but probably yellowish or gray in the first case and purple in the second. Similarly with the bitter drink. Do not, then, confound the sensation with the physical object. Unless they are kept rigorously separated, confusion results.

Adaptation. The term **adaptation,** used without a qualifier, refers to sensory adaptation. It may be defined as a progressive change in the sensitivity of receptors due to the conditions of stimulation. Changes of sensitivity in turn may lead to a change in quality because certain parts of a system are more responsive than others. Figure 6.2 shows an action-potential record of the adaptation of a photoreceptor under a constant stimulus. Note the decreasing frequency of response. A good example of visual adaptation is found upon going into a movie theater in the daytime. At first you can see nothing, because you are adapted to daylight — a very high level of illumination. After a few moments objects become visible and you are able to make your way to a seat without trouble, for you are becoming adapted to a new level of illumination. When you again go outdoors this is obvious by the unpleasantly intense stimulation which greets you at the door. Another example is found in the adaptation of the pressure receptors to rings and spectacles. One simply ceases to feel them after a short time. Changes in quality with adaptation sometimes occur in smells, particularly in disagreeable ones.

In order for adaptation to proceed with any regularity, it is necessary that stimulation be constant. If you stare at a small patch of color without moving your eyes (an almost impossible feat), it will disappear, i.e., adaptation will be complete. The reason small objects do not disappear in the normal course of events is that the eyes are almost constantly moving and stimulation thus varies. It requires a laboratory situation to demonstrate local adaptation of small areas. Adaptation to a general level of stimulation is much more usual in the nonlaboratory situation. All the visual receptors become more sensitive as daylight fades and the general adaptation is not disturbed by moving the eyes because this does not change the conditions of stimulation. We shall soon study adaptation as it occurs in each sense, but it may be well to point out here that most senses adapt, even pain, if the rule of constancy of stimulation is followed. A few senses show some peculiarities in adaptation. One of the senses which shows very little adaptation, for all practical purposes, is audition. The ticking of the alarm clock is just as audible after two hours as it was when you sat down beside it. You may not hear it every moment, but if you wish to discover whether it is still running, you need only turn your attention to it and you can hear it ticking. This is called **attentional adaptation,** for it involves attention only, not actual

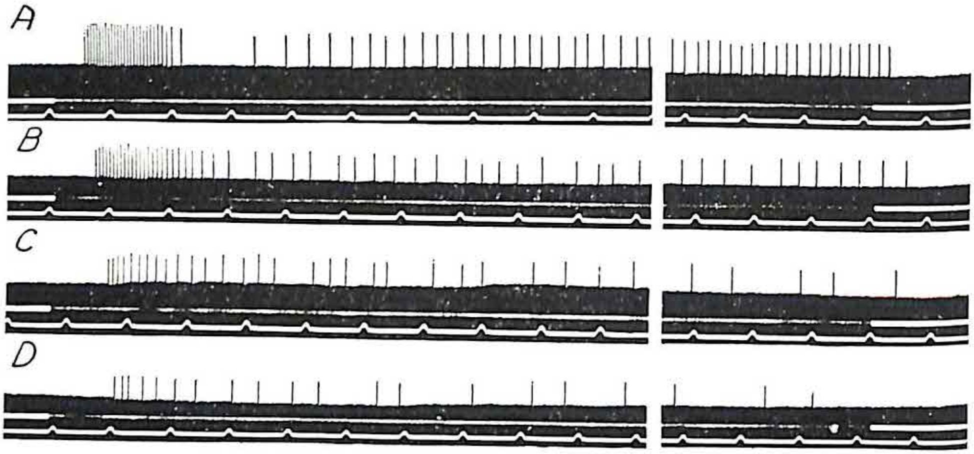


FIG. 6.2. Action potential record of adaptation of a photoreceptor — decreasing frequency of response with continuing stimulation at four levels of intensity. (After Hartline and Graham.[2])

change in the level of response of the receptors. In sensory adaptation you cannot restore the original sensation merely by turning your attention to it. The senses vary, then, in the degree to which they show adaptation and it is a very important aspect of individual sensitivity. In vision and in olfaction, control of adaptation is one of the most important variables in an experiment.

Role of CNS in Sensation. We have already indicated to some extent the role which the central nervous system plays in sensation. In connection with each sense we shall outline schematically the afferent pathways by which each sends its impulses to the highest station in the CNS. It remains here to indicate some generalities which can be expected of all or most senses. As nearly as we can tell at present, sensation is dependent upon excitation's reaching the cortex, although it is possible that some vague awareness may arise subcortically. Reflex responses are possible at the spinal level, but the individual feels nothing if his spinal cord is cut and the impulses cannot reach higher levels. A man who loses his occipital cortex is blind, even though reflex responses to light may be present. It is the patterning of excitation in the primary sensory areas of the cortex that is responsible for sensation. Response can occur below this level, but probably not sensation. In lower animals there appears to be some awareness of dark and light when the cortex is removed, but even here we find no response to more complex patterns. We can feel fairly confident that cortical patterning is necessary for sensation, whereas perception, as we have defined it, rests upon the elaboration of this excitation in neighboring areas, with the relating of this sensory experience to memories of other similar ones, the previous responses and their effects.

Suggested Readings

1. Boring, E. G., "Sensation and Perception," *Sensation and Perception in the History of Experimental Psychology*. New York: Appleton-Century, 1942, Chap. 1, pp. 3–52.
2. Boring, E. G., *The Physical Dimensions of Consciousness*. New York: Appleton-Century, 1933.
3. Boring, E. G., Langfeld, H. S., and Weld, H. P., "Sensation and Psychological Measurement," *Foundations of Psychology*. New York: Wiley, 1948, Chap. 11, pp. 250–268.

7 | *Pressure and Temperature*

Introduction. In this book we have reversed the usual order in which the senses are presented, as many will recognize. We have done so for two reasons, one psychological and one pedagogical. First, the student is usually so impressed with the " major senses " of vision and audition that the contributions of the so-called " minor senses " are neglected since the available facts are not so overwhelming in these latter fields and, therefore, by contrast, appear insignificant. Second, it is easier to learn some basic principles of sensation, and to develop some understanding of how the senses operate, in areas where the known facts are not so many nor so detailed as to be confusing.

Congruent with our opinion that the " minor senses " are of great importance in the behavior of organisms, we shall point out some of the ways in which they do contribute to our behavior, confining the discussion in this chapter to **somesthesis** and, particularly, to pressure and temperature. Somesthesis is a convenient word which refers to body sensibility — pressure, pain, warmth, and cold, both in the skin and in deeper tissues and viscera — but excludes **kinesthesis,** which refers to sensibility in muscles, tendons, and joints. The discussion of pain creates some special problems, so it is treated in a chapter by itself, except in so far as it must be considered in a general discussion of somesthesis. We shall also have occasion to use the word **cutaneous** in referring to some somesthetic sense data — it means the skin senses, rather than deeper ones of the same name.

The somesthetic senses can be considered to serve three functions: (1) they provide certain kinds of information about the world outside us, and this information is different, as we shall see, from that provided by vision or audition; (2) they provide most of our useful information about the world inside us and are thus important in the perception of the self and the body image; and (3) they are a vital link in many drives and motives.

The information which the somesthetic and kinesthetic senses provide about the world around us supplements in many ways the information gained through merely seeing an object or hearing a sound. Texture of surfaces, hardness or softness of objects, tactual and kinesthetic shape all contribute a good deal to our knowledge of how to behave toward objects, and these cannot be known visually, except through previous somesthetic and kinesthetic experience. It should be impressive to the psychology student to learn that the ultimate definition of one of the basic terms in physics, force, is essentially based upon somesthesis and kinesthesis, for it means that which you feel when you push against something, or something pushes you. Again, one of the ways in which you recognize metals is through cutaneous sensitivity — metallic objects feel colder than those of wood or paper. Conversely, one aspect of recognition of wool is its warm feel, whereas nylon feels cold. Examples by the score will occur to anyone if he attends to these sensations during the course of a day. But if normal examples do not suffice, consider the cases of Helen Keller and Laura Bridgman, both of them deaf and blind from a very early age. They were able to learn to utilize the information provided by somesthesis and kinesthesis * and upon this basis alone could deal with people, objects, and symbols. Helen Keller graduated from Radcliffe College, Laura Bridgman did sewing for profit! Neither of these women had abnormally high sensitivity in any of their remaining senses.

It would be hard to overestimate the contribution of somesthesis to the perception and knowledge of the self. The cutaneous senses, even more than vision, define the limits of the self — one's skin, even when he's sitting on it and can't see it, is part of the self; the chair is not. The rather meager knowledge that most of us have about the part of us inside the skin comes from somesthesis and kinesthesis and, of course, our knowledge that something is wrong is based upon pressure and pain.

In the above paragraph the emphasis was upon information alone. But there is the third function of somesthesis, namely, the part played in drives. This will be discussed more fully in the appropriate chapters, so that we will mention here only a few of the more obvious relationships. One important aspect of hunger, for many people at least, is a pressure-pain pattern in the stomach. Thirst, as perceived, depends in part upon " dryness " in the mouth and throat, which is based upon somesthetic sensations. The very compelling drive to empty the rectum is based upon pressure in the rectum itself. Pain plays such an important role in driven behavior that further discussion of it must be deferred to Chapter 8.

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* Helen Keller also had normal smell and taste, and used smell as her only distance sense. Laura Bridgman had very poor sensitivity in both these senses.

The Dissociation of the Somesthetic Senses. In the foregoing discussion we have assumed that the four somesthetic senses are separate or dissociated. This is contrary to popular opinion and, indeed, scientific opinion many years ago held the view that general bodily sensation was a single sense and that pain was not a sense in itself, but occurred upon the excessive stimulation of any other sense. The evidence for the separateness of the somesthetic senses is overwhelming, however. Pain does not result from mere intense stimulation of some other sense — the most intense pressure sensations do *not* become painful, they remain as intense pressure. But the stimulus which arouses intense pressure ordinarily also stimulates the pain receptors. The available evidence serves not only to confirm pain as a separate sense, but also to establish pressure, warmth, and cold as separate from pain and from one another. The lines of evidence are as follows:

(1) Distribution of sensitivity: the skin is not equally sensitive throughout its area, and the areas of maximum sensitivity to pressure (for example, the finger tips) do not show the lowest thresholds for warmth or cold or pain.

(2) Interference with nerve function: local anesthesia, nerve blocks,* and anesthesia applied to the nerve itself result in the disappearance of the four cutaneous senses at different times and in different orders.

(3) Electrical excitability: the four cutaneous senses have reliably different excitation times.

(4) Spinal cord surgery: an operation for the relief of unbearable pain involves cutting the tracts in the spinal cord through which pain impulses travel to higher centers. Sometimes pain is eliminated with little disturbance of sensations of pressure, cold, or warmth.

(5) Pathology: in various pathological conditions one sense department may be lost while others remain intact. Pain alone may be lost, or cold only, or pressure, or warmth. A disease of the spinal cord, syringomyelia, frequently results in the loss of the sense of pain first. There are also cases on record of complete **congenital analgesia,** with the remaining senses normal.

The evidence establishes four somesthetic senses: pressure, pain, cold, and warmth. It does not, however, enable us to decide whether cutaneous, deep, and visceral sensitivity in any one of these categories belongs to a single sense, or two, or three. This problem is more difficult, and what evidence we have will be discussed under each sense.

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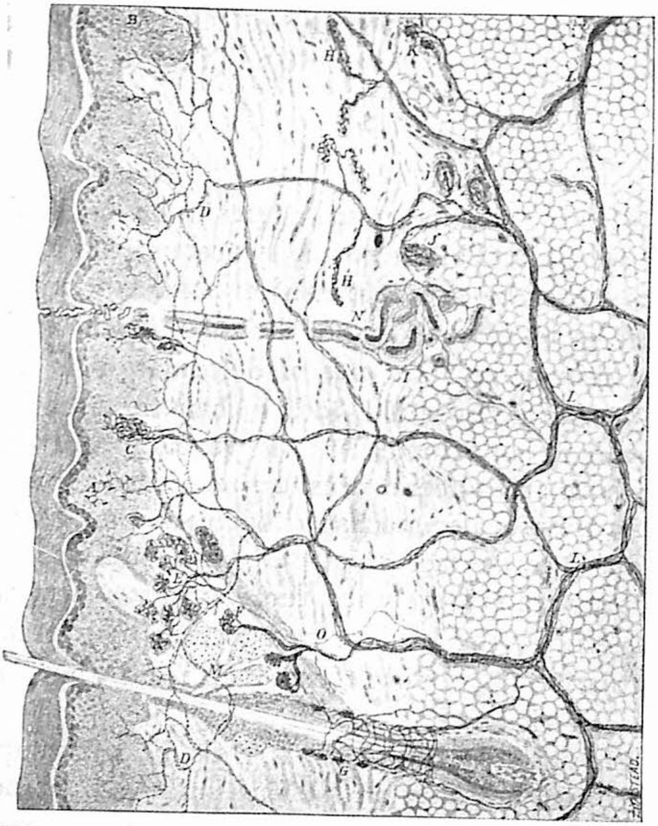
* Pressure on a nerve will interfere with its functioning, as exemplified in hitting one's "funny bone."

PRESSURE

Stimulus. The stimulus for pressure now appears to be mechanical deformation of the receptor. This is normally accomplished for the cutaneous receptors either by the deformation attendant upon contact with some object, or by the bending of the small hairs which are scattered over most skin areas. The rate of deformation is the important variable in attaining threshold, rather than the area involved, as far as the skin is concerned. This is probably not entirely true for deep pressure and visceral pressure receptors, though the adequate stimulus for these is also mechanical deformation. Deep pressure is normally aroused by contact which deforms tissues deeper than the skin; visceral pressure is largely the result of distension, as in the intestines by gas and in the bladder and rectum by normal filling. Inadequate stimulation of pressure receptors is readily accomplished by means of an electric current.

Neuroanatomy and Physiology. The body is, thus, liberally supplied with pressure receptors, not only on the skin surface, but also in depth and in the lining of the various body cavities. We do not, however, find one single kind of receptor, but several. Figure 7.1 shows a composite diagram of the skin and its underlying tissues. It is very complicated and the function of many of the structures is not clear. Cutaneous pressure in hairy regions (which includes most of the body surface) has one fairly well recognized receptor, the basket-like nerve ending around the hair follicle (G in Fig. 7.1), which may be aroused by slight bending of the hair itself. It is possible that there are other kinds of pressure receptors here. In the hairless regions, such as the lips and finger tips, **Meissner's corpuscle** is believed to be the pressure receptor, largely on the basis of distribution. The receptors for deep pressure and visceral pressure are not agreed upon. There are suggestions that some free nerve endings may also subserve pressure sensation.

The nerve fibers of the pressure system are the large ones in an afferent nerve. They run with the fibers for all other somesthetic senses from a given bodily area to the spinal cord, where they are sorted into tracts according to sense **modalities.** The pressure fibers from below the head ascend in the ventral portion of the spinal cord of the opposite side (cf. Fig. 7.2) to two of the posterior nuclei of the thalamus; fibers from the head and face are carried largely in



A – Merkel's discs
B – Free nerve endings
C – Meissner's corpuscles
D – Pain fibers
E – Krause's end bulbs
F – Ruffini's endings
G – Nerve endings around hair follicles
H – Ruffini's endings
I – Sympathetic fibers innervating sweat glands
J – Pacinian corpuscles
K – Golgi-Mazzoni endings
L – Nerve trunks
M – Sebaceous gland
N – Sweat gland
O – Sympathetic fibers to erector pili muscle

FIG. 7.1. A composite diagram showing the receptors in the skin and underlying tissues. (From Wollard, Weddell, and Harpman.[17])

cranial nerve V, the trigeminal, and terminate in the same thalamic nuclei. Here new fibers arise which project to the **post-central gyrus** of the parietal lobe, the great somesthetic receiving area of the cerebral cortex. In this area somesthetic impulses are once again sorted out into a topographical pattern. A kind of reverse body image is spread out on this cortical strip, with the genitals and toes at the top and the face at the bottom. Electrical stimulation of the arm area, for example, will give rise to sensations localized in the arm of the opposite side. But even here functional organization in terms of particular senses is not wholly lost, for with cortical damage different senses may be selectively disturbed.

The way in which each spinal nerve distributes its fibers to the periphery is of interest. Figure 7.3 shows most of the **dermatomes** of man. A dermatome may be defined as the skin area supplied with afferent fibers by a single posterior root of the spinal cord. We thus have a dermatome for each spinal nerve except the first cervical nerve. There are 8 cervical (C), 12 thoracic (T), 7 lumbar (L), 4 sacral (S), and 1 coccygeal nerve. The dermatome is named for its spinal nerve

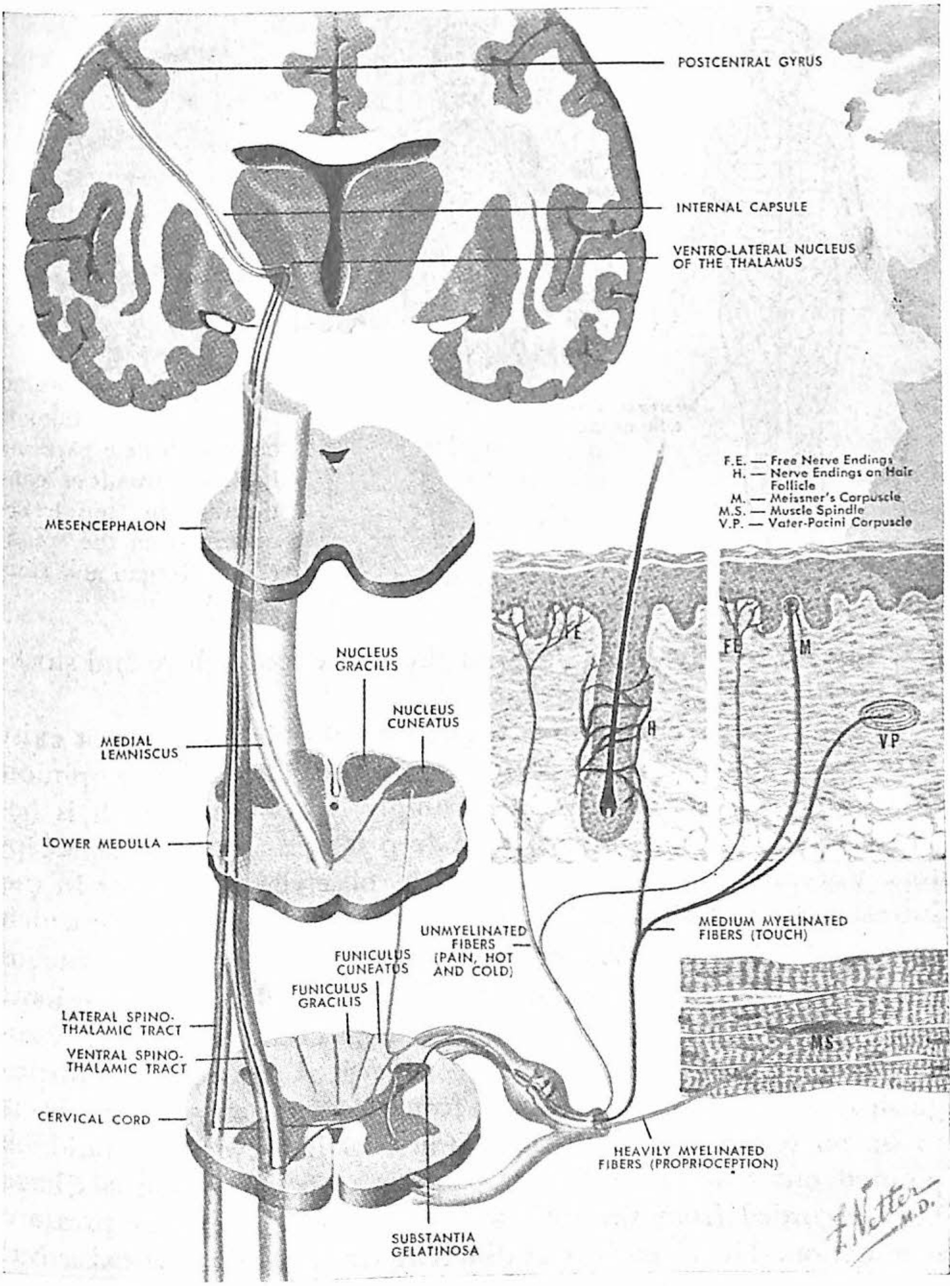

POSTCENTRAL GYRUS
INTERNAL CAPSULE
VENTRO-LATERAL NUCLEUS OF THE THALAMUS
F.E. — Free Nerve Endings
H. — Nerve Endings on Hair Follicle
M. — Meissner's Corpuscle
M.S. — Muscle Spindle
V.P. — Vater-Pacini Corpuscle
MESENCEPHALON
FE
FE
H
M
VP
NUCLEUS GRACILIS
NUCLEUS CUNEATUS
MEDIAL LEMNISCUS
LOWER MEDULLA
MEDIUM MYELINATED FIBERS (TOUCH)
UNMYELINATED FIBERS (PAIN, HOT AND COLD)
FUNICULUS CUNEATUS
FUNICULUS GRACILIS
MS
LATERAL SPINO-THALAMIC TRACT
VENTRAL SPINO-THALAMIC TRACT
CERVICAL CORD
HEAVILY MYELINATED FIBERS (PROPRIOCEPTION)
SUBSTANTIA GELATINOSA
F. Netter M.D.

FIG. 7.2. Diagram of the somesthetic tracts from the body. (From Netter.[18])

and it is very convenient to be able to refer to anesthesia of dermatomes L2 and 3, say, in discussion of clinical cases. In abnormal psychology, one of the important criteria for deciding whether a particular case of anesthesia is organic or **hysterical** is based upon this concept of dermatomes. Typically, organic anesthesia is restricted to dermatome outlines whereas hysterical anesthesia usually is not, but

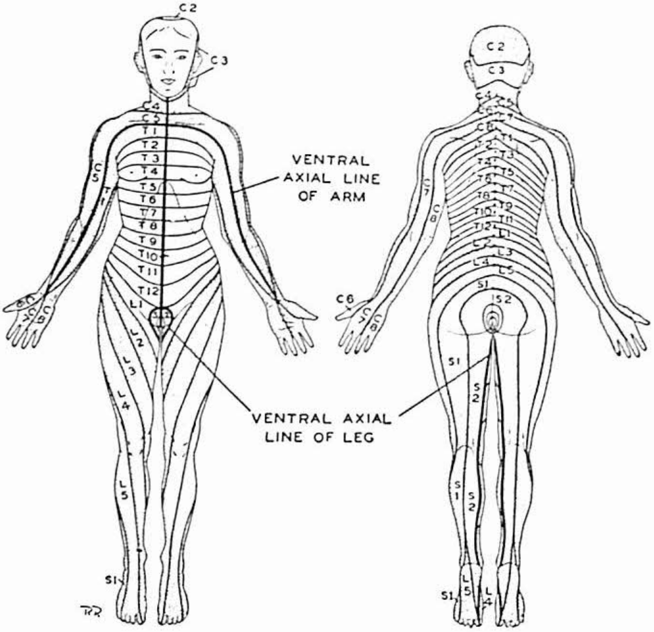


FIG. 7.3. Dermatome chart of the human body with new patterns in the extremities continuing in serial sequence from the spine. (From Keegan and Garrett.[19])

tends to involve the whole hand or the whole foot (glove and stocking anesthesias).

Psychological Dimensions. Unanimity of opinion does not exist as to how many qualities of pressure there are. The majority opinion favors one quality, usually called simply " pressure," which is believed to occur with stimulation of deep as well as cutaneous receptors. Visceral pressures are described by observers competent in the **introspective technique** as " duller " than cutaneous pressures, which are called " bright." Whether this perceptual difference is due to poorer localization of visceral sensations and different concomitant sensations or is a genuine qualitative difference is not known. Pressure is sometimes called " contact " when it is cutaneous, to distinguish it from the pressure arising from deeper tissues. There seems to be no qualitative justification for this. However, it should be pointed out that two different patterns of action potentials have been recorded from the cat's toe pad with two sorts of pressure stimulation. The possibility of different kinds of pressures exists but it is not yet clear how this applies to human beings.

That pressures vary in intensity is obvious. The flick of a single hair on one's arm is readily perceptible but the resting of a heavy object on the arm gives rise to a more intense sensation of pressure. Thresholds are often obtained by using a calibrated series of hairs — horsehairs of various lengths and thicknesses calibrated on a sensitive balance. Figure 7.4 shows a mechanical apparatus which accomplishes the same result more easily.

The absolute threshold for pressure varies considerably over the surface of the body. Table 7-1 shows some representative thresholds. It will be seen that the lowest thresholds occur on the lips and finger tips, the highest thresholds on the back. The actual value of the absolute threshold in any area depends, as you would expect, upon the method used in the determination. Using a well-controlled method, one investigator found that the threshold on the thumb may be stated as .026 ergs.[1]

Table 7-1. Some Representative Thresholds for Cutaneous Pressure in Various Regions

[After Woodworth and Schlosberg (13)]

Area	Threshold (gm. per mm.^{2})
Tip of tongue	2
Tip of finger	3
Back of finger	5
Front of forearm	8
Back of hand	12
Calf	16
Abdomen	26
Back of forearm	33
Loin	48
Thick parts of sole	250

The differential thresholds for pressure also show variation with the area investigated, the smallest DL's occurring in those areas having the smallest RL's. The Weber fraction shows, as usual, some variation, especially at the extremes. Figure 7.5 plots the Weber fraction as a function of intensity level for 1 to 60 grams.

The size of both the absolute and differential thresholds is related to the number of pressure " spots " found in a given area. If the skin surface is explored with a weak stimulus of small diameter, it is discovered not to be uniformly sensitive. Pressure is felt in spots, and the spots occur more frequently and are closer together in regions of low thresholds. These spots are found just to the side of the hairs away from which they bend, (the ' windward ' side) in hairy regions, directly over the basket endings around the hair follicles. As previously mentioned, Meissner's corpuscles occur with about the same frequency as pressure spots in the hairless regions. Figure 7.6 shows the relation between stimulus intensity and number of spots responding. The more intense the stimulus the more spots are called into play.

The extent of cutaneous pressure is correlated rather closely with the extent of the stimulus, particularly in those areas where localization is sharp. Nothing is known about the extent of other pressure sensations. Duration is, of course, related to duration of the stimulus, within the limits set by adaptation.

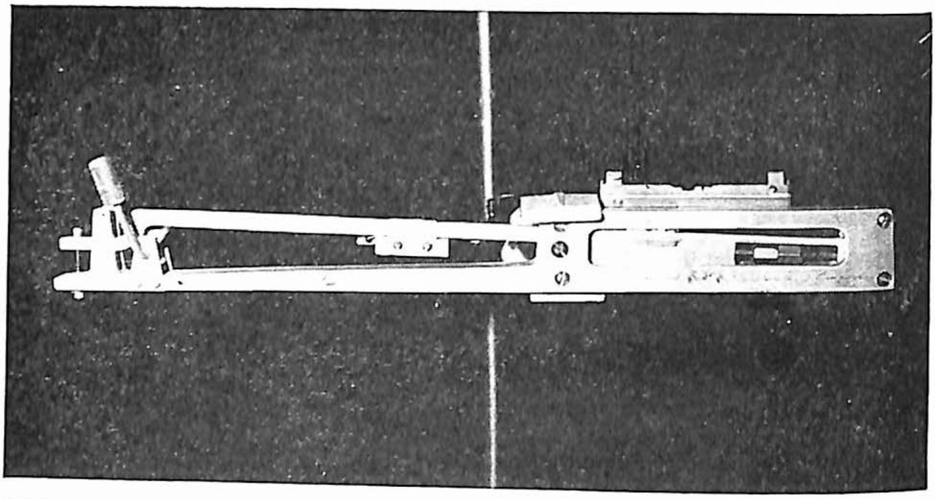


FIG. 7-4. The UCLA esthesiometer. The weight on the needle point is governed by the position of the rider on the lever. The handle raises and lowers the needle.

Adaptation. Adaptation occurs in both cutaneous and deep pressure. Data are not available for visceral pressure. Adaptation is readily observable outside the laboratory — we cease to feel rings and glasses, unless we wiggle and thereby change the stimulus, and even clothing ceases to be felt when one is still. Adaptation time increases as weight increases but decreases as the area stimulated increases. Table 7-2 illustrates this principle. It is often stated that deep pressure has a longer adaptation time than cutaneous pressure because of the fact that heavy weights, stimulating deep pressure, require a longer time for adaptation than do lighter weights but the fact that more intense stimuli do, in general, take longer to adapt, renders further research necessary. According to one study, much supposed adaptation is nothing more than the cessation of the adequate stimulus at the receptor level. It takes some time for the compression of tissues to cease, so that the actual stimulation of the receptor continues to change after the external stimulus has become constant.[2]

After-sensations do exist in pressure and are positive, that is, similar to the original sensation. One is frequently aware of them after removal of a ring when one does not miss it because one can feel it. But it is obvious that some change of stimulus is occurring, in that compressed tissues are released and begin to expand. How much of the phenomenon of positive after-sensations is due to changes in receptor deformation and how much to genuine after-images is unknown.

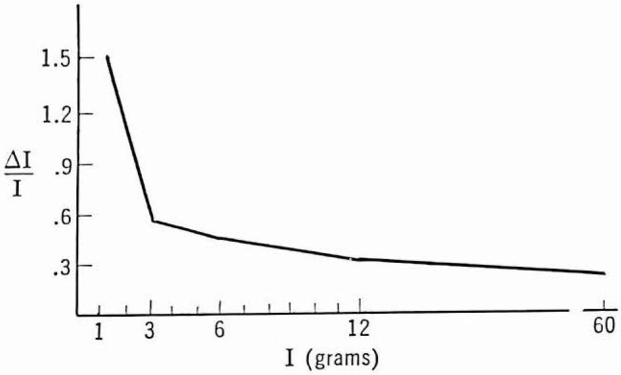


FIG. 7.5. Variation of the Weber fraction as a function of stimulus intensity. Stimulus diameter .1 mm. (After Schriever.[20])

Localization. Localization is an important aspect of pressure, for pressure is essentially a "space sense" like vision. In both these senses, the spatial patterning is the most significant aspect. Localization of pressures, together with kinesthesis and vision, is basic to the development of our concepts of space and position. Accuracy of localization varies both with region and with method employed. Table 7-3 shows the accuracy of localization in various regions. Accuracy is greatest in those areas having the greatest sensitivity and the greatest density of receptors, although it is probably the pattern of excitation, rather than isolated receptors which determine the perception. Although accuracy of localization of cutaneous pressures is reasonably

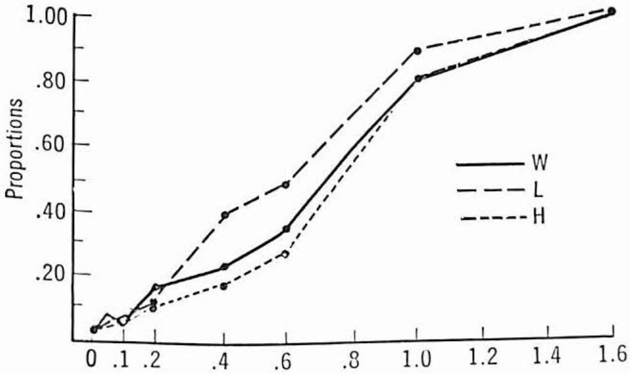


FIG. 7.6. Percent of spots responding with various intensities of stimulus (1 sq. cm. area). (After Guilford and Lovewell.[21])

Table 7-2. Pressure Adaptation Times for Various Weights and Areas

(Adaptation time in seconds)
[After Zigler, (14)]

	Stimulus				
Region	Weight in Milligrams				
	50	*100*	*500*	*1000*	*2000*
Back of hand	2.4	3.8	6.0	6.7	9.5
Forearm	2.3	3.3	4.9	5.6	7.8
Forehead	5.1	6.2	10.0	10.4	16.0
Cheek	5.7	6.4	11.6	13.5	19.7

	Stimulus				
Region	Diameter in Millimeters				
	5	*10*	*15*	*20*	*25*
Forehead	18.9	16.1	14.6	12.3	9.9
Cheek	21.2	16.4	14.4	12.2	10.3

good in many areas, it has been found that pain is a little better localized and that the two in conjunction yield even better results.

Localization of visceral pressure is poor, at least in most people. Visceral pressures tend to be rather diffuse, probably partially because of the nature of the usual stimulus, but also possibly because one has never learned to localize them. In addition, visceral sensations are often " referred " (see Chap. 8) and are perceived as coming from some point on the body surface. Recent studies have shown that visceral afferent fibers have many connections to the postcentral gyrus of the cortex, so that one would expect, on anatomical grounds, rather accurate somesthetic perceptions of visceral events. It has been suggested that these impulses are normally partially blocked subcortically by the more frequent impulses from exteroceptors. In those neurotic states in which there is unusual awareness of visceral events, it may be that cortical activity has modified the subcortical block.

Interaction. Interactions occur both between various pressure stimuli and between several somesthetic stimuli of different kinds. Summation is an important characteristic which, as Chapter 3 explained, is a function of the synapse. Spatial summation, the integration of spatially disparate impulses, does occur in the pressure sense. Two subthreshold stimuli will, if presented simultaneously at neigh-

Table 7-3. Accuracy of Localization of Pressures for Various Bodily Regions

[From Woodworth and Schlosberg (13)]

Region	Error (cm.)
Forehead	.63
Lip	.10
Chin	.54
Forearm (volar)	.85
Back of hand	.65
Palm	.43
Fingertip (volar)	.10
Thigh	1.60

boring points on the skin surface, be perceived, whereas neither one is sufficient alone.

Tickle is a peculiar effect which results from a temporal and spatial pattern of pressures. Pressure is also one factor in perceptions of wetness or oiliness of objects or of dryness in the throat. Perception of vibration results from successive stimulation of pressure receptors.

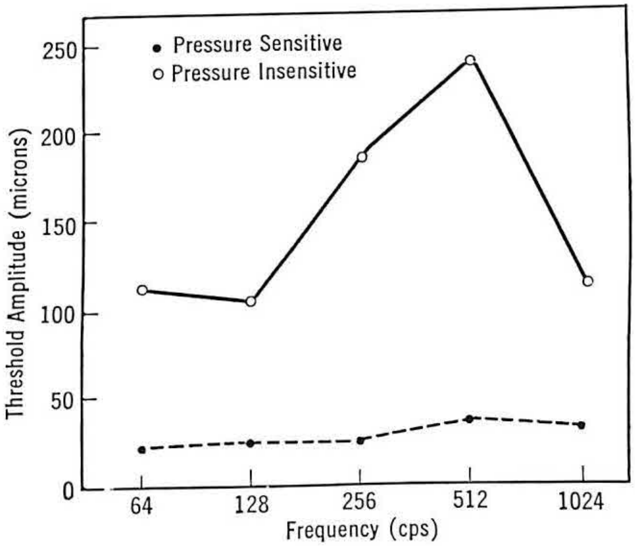


FIG. 7.7. The vibration thresholds for pressure-sensitive (dotted line) and pressure-insensitive (solid line) areas. (After Geldard.[22])

Figure 7.7 shows the response of the receptors in areas with high and low thresholds for pressure to both amplitude and frequency of vibration. Table 7-4 shows the sensitivity of various bodily areas to vibration at 120 cycles per second. It can readily be seen that the regional sensitivities are approximately the same as those obtained

Table 7-4. Vibratory Thresholds in Microns of Motion at 120 Cycles per Second

[Adapted from Gregg, (15)]

Region	Threshold (10^{-4} cm.)
Forehead	6.16
Shoulder	7.40
Forearm	6.90
Wrist	0.780
Fingertip (first)	0.370
Trunk, Anterior (at T2)	2.96
Trunk, Posterior (at T2)	1.47
Thigh (L2)	8.88
Calf, distal side	2.96
First toe, tip	0.838
Sacrum	12.3

by other methods. Sensitivity to vibration is highest in adolescents and decreases with age, particularly after 50 years of age.

Studies of **stereognosis,** or tactual form perceptions, have shown that it is extremely poor. The clearest — or better, the least unclear — perception of tactual form occurs with figures having unbroken outlines. Acute angles in a figure are also important landmarks to its recognition. As you would expect, recognition is best in areas showing greatest sensitivity and best localization.

It is apparent from experimental work on perception of tactual form, from a study of chimpanzees reared in darkness and from reports of cases of congenital cataract where eyesight has been restored late in life, that perception of nonvisual form (largely tactual and kinesthetic) is not the same thing as the perception of visual form. Someone who has first learned nonvisual forms does not always find it easy to identify visual forms upon first sight, that is to say, tactual five-pointedness is not the same thing to the organism as visual five-pointedness. And normal people have great difficulty in recognizing tactual shapes unless they enlist the aid of visual imagery.

The armed forces have sponsored research upon recognition of aircraft-type control handles by " feel," both tactual and kinesthetic cues being called into play. Accuracy and speed of recognition varied considerably, depending upon the shape of handle used, but in some cases errors were small (12 percent),[3] suggesting possible use of this sort of sensory cue to relieve somewhat the overburdening of vision in modern aircraft (or automobiles, for that matter!).

TEMPERATURE

We discuss warmth and cold together because of similarities in the stimulus and in the proper experimental controls. It must be kept constantly in mind, however, that the two senses are separate and distinct — it is only for convenience's sake that they are placed together. The term " warmth " sometimes gives trouble; warmth is the proper term for the sense department and for the sensation, not heat. The two terms are not interchangeable. Heat is a more complex perception than warmth and normally results from the simultaneous stimulation of warmth receptors and pain receptors.

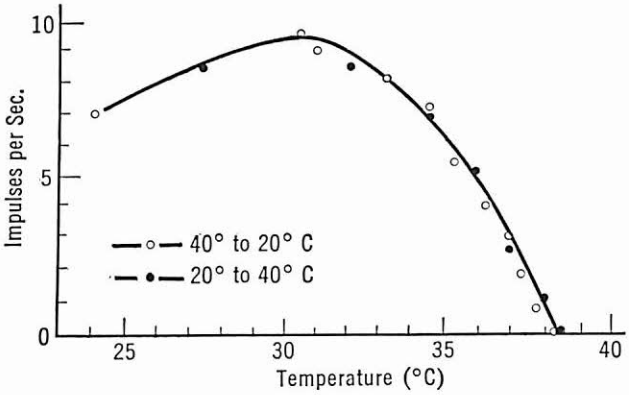


FIG. 7.8. Dependence of frequency of action potentials upon temperature in a single fiber of the lingual nerve of the cat. (Hensel and Zotterman.[4])

Stimulus. The adequate stimulus for both cold and warmth has turned out to be an absolute temperature, not the rate of change as formerly thought. Some very ingenious research, in which the temperature beneath the skin has been measured, has proved that action potentials in cold fibers are controlled by the actual receptor temperature, not by temperature gradient or change.[4] Figure 7.8 shows the dependence of frequency in the aciton potentials in a single nerve fiber upon absolute temperature. Frequency increases rapidly as the temperature falls below body temperature. It should be noted that there is some response even at temperatures above blood temperature (35° to 37° C in the cat) — the dot at about 39°, for example. The total frequency of impulses in the cold fibers of the entire nerve (*not* the single fiber, however), over the range of 20° to 40° C, acts as a very nearly rectilinear thermometer-function, the number of impulses per second representing rather precisely the temperature of the stimulus. With very rapid cooling of the receptors, the frequency of impulses can be forced as high as 140 per second, but it falls rapidly towards a constant end-point appropriate to the final temperature. With very rapid warming from low temperatures toward blood temperature, the impulses disappear, but return soon and take up the frequency proper to the final temperature. These action potentials have been followed for as long as 70 minutes with various stimulating temperatures and they are found to persist unchanged throughout the entire period, showing that the temperature receptors respond to temperature as such, for any changes which occur in the temperature of the receptor are over within a very few minutes.[5] It has also been shown that stimulation applied from underneath the receptor, instead of on top, results in both similar action potentials and in similar sensations — the temperature gradient is unimportant.[6]

Table 7-5. Thresholds for Warmth for Large Areas

[From data of Maréchaux and Schäfer, (7)]

Average skin temperature for whole body

	S1	*S2*	*S3*	
At beginning of Experiment	31·5	30.0	30.2	slow temp. rise (.05° per minute)
At arousal of Warmth	34·3	34·3	33·6	
At beginning of Experiment	31·7	30·9	32.0	rapid temp. rise (.1–.2° per minute)
At arousal of Warmth	34·5	33·9	35·6	

Another recent study showed that warmth behaves similarly, even under conditions like those of daily life where the entire body is subjected to changes of external temperature. Table 7-5 shows a summary of part of these results for three subjects. The arousal of warmth occurs at approximately the same skin temperature regardless of whether the temperature rise in the climate room was fast or slow. Even the presence of a long fore period does not change the results significantly. Sensations of warmth arise at an average skin temperature of 34° to 35° C. Various parts of the body have their own critical temperatures, but the point at which warmth or cold sensations arise is relatively constant.[7]

Neuroanatomy and Physiology. The receptors for cold and warmth are not definitely known. They are assumed to be the free nerve endings of their respective nerve fibers. The excitation data indicate that the two kinds of receptors are located at slightly different depths in the skin, with the depth of the warmth receptors being somewhat more variable than that of the cold receptors. The afferent fibers run in the lateral portions of the opposite side of the spinal cord, near the pain fibers (see Fig. 7.2), to one of the posterior nuclei of the thalamus. From here, projection fibers arise that terminate in the postcentral gyrus of the cerebral cortex. Temperature impulses from the face travel via cranial nerve V, the trigeminus. In the cortex, all somesthetic sensations from one bodily area are grouped together, the cortex being organized according to area (topographically) rather than according to sense modality (functionally).

Psychological Dimensions. In each sense there is but one cutaneous quality, cold for the cold sense and warmth for the warmth sense. Temperature senses in the viscera are not aroused in the normal course of events. Civilization has introduced us to hot and cold foods, but primitive man, without refrigeration or even fire and living in warm climates, certainly knew little of these pleasures. Warmth and cold can, of course, be elicited in the mouth and esophagus. It is sometimes said that visceral warmth and cold are duller than their cutaneous counterparts qualitatively but, again, we must say that a possible explanation for this lies in poorer localization and different concomitant sensations.

Intensity as a dimension of cold and warmth is readily observable. In general, temperatures above normal skin temperature give rise to sensations of warmth and these sensations become more intense as the temperature of the stimulus rises. Conversely, temperatures below normal skin temperatures do, in general, give rise to sensations of cold which become more intense as the temperature falls. However, there are two important phenomena which modify this relationship.

The first limitation is imposed by the effective terminal limens, that point at which the stimulus temperature arouses pain. For the warmth sense, this lies at 50° C, at which point pain enters the picture. For the cold sense, the effective terminal threshold is 18° C. Temperature sensations mixed with pain can be evoked with stimulus temperatures above and below these points, but there is some indication that the receptors fail to respond at all at temperatures above 70° C and below —10° C. Tissue damage occurs well before these extremes are reached.

The second limitation is imposed by the inadequate stimulation of both warmth and cold by temperatures within the range of the adequate stimulus for the other one, that is, arousal of warmth by a stimulus below skin temperature and arousal of cold by a stimulus above skin temperature. These effects are called " paradoxical " cold and warmth. Paradoxical cold is the more easily observed of the two and frequently occurs during mapping of an area for warm "spots" with a small stimulus. Paradoxical cold occurs between about 43° C and 50° C, when the cold receptors are stimulated inadequately. Paradoxical warmth occurs between about 25° and 31° C, when the warmth receptors are stimulated inadequately.

There is little agreement as to the absolute threshold for either sense, partly because of difference in experimental method, partly because adaptation effects are significant here, as in vision. In work on action potentials, it was found that some cold fibers (whose receptors had low thresholds) responded to a drop of 0.1° C below the neutral point (physiological zero), whereas others required as much as several degrees.[8] The study of temperature changes for the total body, previously mentioned, showed that an increase of less than .2° per minute is effective in inducing sensations of warmth. For small areas, the absolute threshold for warmth is reported to be very much smaller than this; for cold, the absolute threshold is of the same order of magnitude. These data agree well with those obtained from action potentials.[9] [10] The difference limen varies considerably with the part of the body and with the point on the scale under consideration. Table 7-6 shows the dependence of the DL upon the temperature of the standard. Weber's law is said not to be valid for the temperature senses but, since the real stimulus at the receptor level is at present unknown, the point cannot profitably be debated.

As with the other cutaneous senses, exploration of the skin with warm or cold stimuli of small size and moderate intensity results in the discovery of "spots" — warm spots and cold spots. The location of these spots is rather reliable — more so for cold than for warmth. There are more cold spots than warm spots and the cold spots are smaller. These spots probably result from pattern of stimulation, rather than stimulation of a single receptor.

Extent, as a psychological dimension, is of some significance. Effects of stimulation are different for small areas and large ones, partly because of the efficient mammalian temperature-regulating mechanisms. Localization, although not precise, is accurate enough to enable one to identify the area stimulated and the approximate extent of skin area involved. With practice, subjects can learn to localize rather well, better in some areas than for pressure. Duration is likewise of interest, but is modified radically, within the usual temperature range encountered, by adaptation.

Adaptation. Adaptation occurs readily in both cold and warmth. The effects are somewhat different for small areas, for larger regions,

Table 7-6. Average Difference Thresholds of the Temperature Senses at Various Temperatures of the Standard

[After Pütter, (16)]

Temperature (°C)	DL(°C)
4	9.2
6	7.0
8	5.1
10	3.8
12	2.8
14	2.0
16	1.5
18	1.15
20	0.95
22	0.80
24	0.70
26	0.60
28	0.50
30	0.60
32	0.70
34	0.80
36	0.90
38	1.00
40	1.10

such as the whole hand, and for the entire body. For the whole body adaptation does occur to temperatures within a few degrees of **physiological zero.** Physiological zero is the narrow range of temperatures which feel indifferent — neither warm nor cold. It is slightly different for each region of the body and varies in each region somewhat, depending upon external temperature, radiation, exercise, and health.

Any wide deviations from physiological zero in the stimulus for the entire body cause " blunting " or partial adaptation; the physiological responses called into play by extreme temperatures over large areas are violent — shivering and sweating — because of the homeostatic temperature-regulating mechanism.

For small stimuli adaptation is complete (even though the impulses in the afferent nerves continue) and the time required for adaptation increases with the area of the stimulus. There are no great differences between warmth and cold in adaptation characteristics. Adaptation to very small areas occurs in 12 to 35 seconds for warm stimuli (43° C),[11] and in 20 to 50 seconds for cold stimuli (11° –16° C),[12] although the variability from spot to spot is large, depending upon the sensitivity of the receptors at the particular spot.

Adaptation of moderate-sized areas shows results in between the previous two. If the fingers are placed in water held at a constant temperature, complete adaptation will occur only between about 25° and 35° C. But adaptation is almost complete between about 10° and 40° C. Adaptation of this sort results in a shift of physiological zero, so that temperatures which previously felt cold will, after adaptation to a still lower temperature, feel warm. For example, after adaptation to 10° C, 12° C feels *warm,* and after adaptation to 40° C, 38° C feels *cool.*

Another after-effect of stimulation is the presence of after-sensations. The after-sensations of both cold and warmth are positive. There is a direct relationship between the intensity of sensation reported for a spot and the duration of these after-sensations, when the original stimulation is held constant at one second. It is probable that the after-sensations are due to continuing stimulation at the receptor, since the temperature of the receptor changes relatively slowly.

Complex Effects. The temperature senses have not been as extensively studied as the other somesthetic senses and we, therefore, know less about them. The evidence as to the presense or absence of spatial summation effects is conflicting and has not yet been resolved. The temperature senses enter into certain perceptual phenomena, such as wetness, in addition to their importance in the regulation of body temperature. The perception of heat, burning heat, and burning cold all require the addition of pain to one of the temperature sensations. It is a curious fact that the perception of heat can often be obtained by the simultaneous stimulation of warmth and cold receptors. It may be that the burning heat one sometimes feels when one puts his hand under the bath tap to test the temperature of the water results from the adequate stimulation of warmth and the inadequate stimulation of cold — paradoxical cold. Form perception in the temperature senses is reported to be nil but, in view of the fact that localization is very poor until much practice is given, this result may be spurious. In addition to functioning as temperature-regulating devices and as exteroceptors giving important information about the external world, warmth and cold are important in the flavor of food and also in other pleasurable experiences, like hot showers or open fires.

Suggested Readings

1. Woodworth, R. S., and Schlosberg, H., *Experimental Psychology,* Rev. Ed. New York: Holt, 1954, pp. 267–296.
2. Jenkins, W. L., *Somesthesis,* S. S. Stevens (Ed.). New York: Wiley, 1951, pp. 1172–1190.
3. Geldard, F. A., *The Human Senses.* New York: Wiley, 1953, pp. 211–232.

8 | *Pain*

Pain is one of the recognized somesthetic senses and is, in some ways, similar to the others in the same group. It, nevertheless, has some peculiarities which place it in a class by itself. In Chapter 2, the concept of equilibrium was developed at some length and it was stated there that much of the behavior of organisms is elicited by noxious stimuli. Noxious stimuli upset the equilibrium of the organism; they are highly motivating in and of themselves and do not require the overlay of learning which most other kinds of stimuli require before they become motivating to an organism. Noxious stimuli typically elicit avertive behavior and assume priority over other sorts of stimuli. For example, the flexion reflex (Chap. 16) is elicited by noxious stimuli and takes precedence over other reflexes; or the amoeba retreats from the irritating chemical and interrupts its feeding. These noxious stimuli, which are so important in motivation and also in emotional behavior, usually are interpreted as painful.

Pain-provoking stimuli are the most important kind of noxious stimuli. There are other stimuli, however, which evoke avertive behavior — bitter substances and high tones, for example; so " noxious " is not equivalent to " painful." The noxious character of stimuli, of course, is derived from the avertive behavior of the organism. There is another aspect which we call " unpleasantness," largely derived from introspective reports from human beings. With lower animals it is possible to study only the avertive behavior. Pain itself, as experienced or felt, can be studied only in human beings. The unpleasantness aspect of stimuli is also limited to work on human beings and is a different concept from pain. Most pains are unpleasant; but the mild pains of carbonated beverages, pepper, wiggling a loose tooth, and scratching an itch may be pleasant. On the other hand, some nonpainful stimuli are unpleasant; such as extremely sweet substances or intense, cloying odors. Since pain has these other aspects which make it different from other senses, it becomes necessary to treat it differently. We, therefore, shall discuss pain first in its sensory aspects and then discuss its motivational and emotional aspects.

Pain-Receptive Structures. Many bodily structures yield pain upon proper stimulation. The skin is particularly well-supplied with pain receptors and we all have experienced the pain occasioned by stimulation of these end-organs as in pricked fingers, minor burns, and stubbed toes. Deeper tissues, lying beneath the skin, are also sometimes involved in these experiences. It is the skin pains which have been most thoroughly investigated, since the skin is more accessible than other structures; so most of our information about the psychophysics of pain is based upon skin pain. Muscles also contain pain receptors; witness the soreness following over-exercise, or after a blow upon a muscle. About muscle and joint pain we know very little. Other deep structures also yield pain. The viscera may be painful under certain circumstances — the pain accompanying indigestion or appendicitis, for example; and pain receptors in the blood vessels may underlie crippling headaches. We are, then, liberally supplied with receptors for pain.

The obvious question which occurs next is whether or not the pains from different structures are identical. We distinguish between red and blue, although we group them both under visual sensations of color. Should we differentiate between visceral pain and skin pain? Are they qualitatively different, or is the difference merely one of location and other concomitant sensations? Skin pain is well-localized and it is very difficult to stimulate it without also stimulating pressure, cold, or warmth. Visceral pain, on the other hand, is diffuse and poorly localized. We cannot at present say whether there is more than one kind of pain, but it seems safest to assume that there are some differences until positive information is available to the contrary.

In the past, the most prevalent terms for differentiation have been " superficial " and " deep " (really meaning outer skin layers versus viscera and deeper layers), or " sharp " and " dull." Sharp pains, which characteristically do not persist, are usually attributed to superficial skin areas. Dull pains, which often persist for longer periods, are usually attributed to receptors lying in deeper tissues or in the viscera.

Stimulus for Pain. The actual stimulus for pain is not known. We know of many things which will evoke pain — cutting or puncturing the skin, applying an electric current, bringing a hot object too close to the skin, putting alcohol on a scratch, and so on — but we do not know what it is about each one of these that actually stimulates the receptor for pain. It has been suggested that the stimulus for pain is anything which causes tissue damage, but this is easily shown to be inadequate by a consideration of the electrical stimulus, which does not damage tissue at intensities sufficient to evoke pain. It is possible also to damage tissue without evoking pain (subpainful sunburn is an example). Finally, in a study of a large number of men who had just received war injuries, no relationship was found between the severity of the wound and the amount of pain it produced. We must conclude that pain has no single adequate stimulus, as do other senses. Mechanical, electrical, thermal, and chemical stimuli all produce pain, apparently adequately; but we do not know what the factor is in each of these which excites the receptor.

Receptors. The receptors for pain are free nerve endings—type 1 in Figure 6.1 since they are neither specialized themselves nor do they have specialized epithelial cells. The nerves subserving pain branch profusely as they enter the general area of their termination, spreading out over a large area (often several square centimeters). The branches lose their myelin sheaths, if they had any, and also the neurilemma, and become very fine, intertwining and overlapping with the terminals of other pain fibers. Any given area of skin is thus innervated by the terminals of a number of nerve fibers. The pain receptors in muscles and in the viscera and blood vessels are also thought to be free nerve endings, although the pain receptors in the viscera must be different in some ways from the more familiar ones in the skin because they seem to be more selective. In abdominal operations under local anesthetic, it has been found that certain of the viscera can be pinched, cut, or burned without evoking pain. But they respond vigorously to distension or pulling. The uterus apparently can be cut and burned without pain, but strong contractions cause great pain. The intestines can also be handled, cut, or burned, but they become very painful upon distension and are easily irritated by chemicals. What the differences are between pain receptors in the viscera and those in the skin are unknown at present.

Neuroanatomy and Physiology. The pain system has, as we saw in Chapter 6, its own receptors, its own nerve fibers, and its own pathways within the central nervous system which we must now examine more closely. The nerve fibers themselves are mostly small myelinated fibers conducting at a rate of 6 to 30 meters per second.[1] These nerve fibers (except from the head) travel to the dorsal roots of the spinal cord where their cell bodies are located. The axons enter the cord and terminate on cell bodies in the posterior horn. The axons arising from the latter cells cross to the opposite side at the same level at which they enter the spinal cord. They there turn upward and run without synapse to the tectum and thalamus.

Pain from the head follows a different course. In common with all sensation from the head, it is carried by cranial nerve V, the trigeminus, but reaches the same tectal and thalamic terminations as pain from the rest of the body. Some pain fibers terminate in a ventral nucleus of the thalamus. From here their course is uncertain. This nucleus of the thalamus has cortical projections to the post-central gyrus, so it is probable that, in part, pain finds its cortical representation with the rest of the somesthetic sensation. This nucleus also has connections to the hypothalamus, which may account for the ease with which pain arouses emotional response.

Pain also finds representation, without thalamic relay, in the frontal part of the old brain. These are the fibers which are interrupted in the frontal lobotomy operation for intractable pain, which apparently removes the insistent quality of the pain. Whether or not these two cortical loci are separately responsible for pain as an informational sense, and pain as emotion-producing, is pure speculation.

Psychological Dimensions. **Quality** of pain has already been mentioned briefly in the discussion of pain from various bodily structures. Within any given general structural division — skin, muscles, tendons and joints, or viscera — there appears to be but one quality, which we call simply pain. As far as difference in quality between pains from various bodily structures is concerned, we have already indicated that we do not have sufficient information to judge. It is very difficult to judge pain quality because of the fact that other senses are almost always aroused at the same time, creating quite different total perceptions; and there also are great differences in the sharpness of localization possible in different areas. Another difficulty which besets experimentation is that the production of pain causes chemical changes in the tissues, which modifies subsequent effects.

Intensity is the most obvious psychological dimension in the sense of pain, but little experimental work has been done upon it except at threshold levels. It is not easy to find subjects who are willing to bear intense pain, nor is it easy to be objective about it even if one is willing to serve as a subject. As in all psychophysics, threshold measurements vary considerably depending, among other things, upon the method used, the area, and the number of subjects used. Table 8-1 gives some representative thresholds for several kinds of stimuli.

Duration of a pain is likewise easily appreciated. A pain may be as brief as a pinprick, or it may last for years. An itch may result from sequential stimulation producing brief pains which are close to threshold.

Table 8-1. Representative Pain Thresholds

Kind of Stimulus	Threshold	Kind of Pain
Mechanical (sharp needle) (7)	1.7 gm. (av.)	cutaneous
Thermal (radiant heat) (8)	182–302 mc./sec./cm.^{2}	cutaneous
Thermal (cold water) (9)	18°C	cutaneous
Electrical (10)	25 volts	cutaneous
Chemical (Potassium chloride) (11)	.12 percent sol.	cutaneous
Ischemia (12)	30–80 contractions	muscular

Extent of pain is not as easy to assess because of the fact that localization is relatively sharp in some areas and very diffuse in others. Nevertheless, one can distinguish between a pinprick and a blow that causes the entire arm to ache, or between a small pocket of gas in the intestine or an all-encompassing stomach ache. These four dimensions — quality, intensity, duration, and extent — enable us to describe pain in the same way that we describe other senses. The total perception is, of course, colored by accompanying sensations. Fears, expectations, and various autonomic and somatic responses make the observation of pain, under both nonlaboratory and laboratory conditions, exceedingly difficult.

Adaptation. Some pain shows adaptation just like most other sensations. At least cutaneous pain adapts. If your arm is immobilized in a plaster cast, and a needle is thrust into it, the pain which arises will gradually lessen and at last disappear. If you move any muscle, or if the stimulating object moves, a change in the position of the needle occurs, and the stimulus is then not constant. Under these circumstances pain will recur; and it will also reappear when the needle is withdrawn. If the stimulus at the receptor is perfectly constant, the receptor adapts. Pains from radiant heat and radiant cold also adapt.

About adaptation of internal pains we do not know. Maybe they adapt under proper circumstances; maybe they do not. Outside the laboratory painful stimuli are rarely constant. One moves, breathes, and digests; all of which disturbs tissue relationships. Our various visceral activities continue more or less uninterruptedly. Perhaps these continuous activities are the factors which induce some of our continuous aches and visceral pains. Perhaps such pains would show adaptation if we could control this activity. At the moment we can say only that some forms of superficial pain show adaptation; some forms of deep pain seem to persist.

Localization. Reference has already been made to the localization of pain, but the problem should be considered in greater detail. As with the other skin senses, if the skin is explored with weak stimuli, pain " spots " will be found scattered here and there among areas of lesser sensitivity. These " spots " cannot result from the stimulation of a single receptor, as in touch, because of the complex spreading and intertwining which occurs among the free nerve endings of pain fibers. Rather, they are thought to correspond to the most sensitive branches of a neuron's terminations.

Localization itself is thought to depend upon the pattern of neural excitation. The prick of a needle at a given point will excite a branch of one neuron strongly, of several others less strongly, and a pattern of excitation results. This pattern is interpreted as a definite point on the skin. Localization is rather accurate on the skin. An average error of localization of 3.6 millimeters for pricks on the back of the hand has been reported — slightly smaller than that for touch.[2] But pains from deeper tissues are not well-localized as far as we know, although they are difficult to investigate. Localization in both muscle and viscera appears to be gross.

Interaction. The experimental evidence for spatial summation effects in pain is conflicting, but it appears that two weak pains close together on the skin (within the two-point limen) summate to give a more intense one.[3] An intense stimulus may induce **hyperalgesia** in a surrounding area, a condition in which the threshold for pain is lowered so that even a light touch may be painful. It also is true that under certain conditions one weak pain may mask another of approximately equal intensity; presumably this is something more than mere distraction. What the relations are among these phenomena and what accounts for them are matters for future research.

An effect of some practical importance is the relationship between pain and muscular tension. Relaxation appears to raise pain thresholds, tension seems to lower them. If you can learn to relax in the dentist's chair, you will feel less pain; tensing your muscles and holding tightly to the chair will make the pain worse.

Pain as a Medical Problem. Headaches are so common a form of pain that they are of general interest. Nearly everyone has one occasionally, some individuals have them frequently and very severely, and others have them almost constantly. There are so many possible causes that it is often difficult to find the responsible one in any given case. They may accompany indigestion, sinus infections, virus infections, or even tenseness and anxiety. One common cause for headaches is distension of the cerebral arteries, and, frequently, it is the aftermath of tension and worry. Administration of a drug which contracts the arteries usually will relieve headaches of this sort. The nerve impulses associated with headache are carried in the pain fibers of cranial nerve V, the trigeminus.

There occur cases of severe chronic pain which often become worse and involve more and more of the body as time goes on. A particular kind of severe pain, an intense burning with certain other symptoms, is called **causalgia.** If unchecked it tends to spread, as from one toe to other toes, and then to the other foot. These long-continued pains (often with no obvious cause) which tend to become worse and to spread are almost certainly not receptor problems. They probably involve **reverberating circuits** in the central nervous system (cf. Chap. 16). Once these are established, frequently by some minor injury, they seem to be self-sustaining and to recruit other neighboring neurons, causing the spread of the pain. If these pains are interrupted by local anesthesia or surgery early in their development, they may not recur. The more severe, widespread, and long-continued the pain, the more damage it does to the organism. Severe pain causes muscular contraction, which may seriously reduce blood supply to an area, thus causing more pain and tissue destruction. Pain also has been shown to interfere with kidney function. Severe pain is avoided by the organism whenever possible but, if avoidance is not possible, it induces physiological changes in the organism of a far-reaching nature and eventually anatomical changes result (cf. Chap. 1: Structure and Function). In addition, emotional behavior is changed. The man in pain, even with only a mild headache, is apt to be irritable or gloomy. The ultimate step in the avoidance of pain is suicide. Many of those people who annually take their own lives appear to be motivated in large part by pain of long standing.

Factors that Reduce Pain. Of interest is the effect of **analgesics** upon pain. An analgesic is a substance which reduces pain without producing unconsciousness. Aspirin is such an analgesic, as are morphine in small doses, codeine, and many other substances. The powerful analgesics, which must be prescribed by a physician and be used with caution, are quite effective in most cases in reducing the severity of pain. Like remedies for seasickness, they are much more effective if administered before pain occurs. The mild analgesics, like aspirin, which we prescribe for ourselves for headaches and mild pains, are apparently effective for such things, but they produce little measurable effect upon the threshold for cutaneous pain. It is possible that we have here an example of difference in response between cutaneous and deep pain.

There are many other factors which decrease our experience of pain. Most of us have had the experience of being injured in some fashion during strenuous activity to which we were attending rather completely and not realizing until later that actual injury had occurred. This is largely a matter of attention; even pains that are more or less continuous and intense can be reduced in apparent intensity if we attend to something else.

Another method of reducing apparent pain is hypnosis. It has been demonstrated many times that hypnosis can entirely eliminate pain in many individuals, sometimes for only short intervals after the hypnotic session, but sometimes for days or even weeks. Probably one of the most significant therapeutic uses of hypnosis is in the control of intractable pain, that is, severe pain of long-standing which cannot be relieved by drugs. Hypnosis has even been used instead of an anesthetic in some major operations and one might ask why it is not so used more frequently. Ideally, it might be preferable to most commonly used general anesthetics because of its safety and because of the absence of unpleasant side-effects. The reason for its infrequent use is to be found, in all probability, in the mores and taboos of our culture. Had ether not been discovered at just the time it was, hypnosis probably would have been employed extensively. Its first successful use in surgery occurred just before the discovery of ether.

Closely related to hypnosis controlled by an operator is self-hypnosis, or autosuggestion. Although it is much more difficult to convince ourselves that a pain does not exist than it is to let someone else convince us, it is possible to indulge in at least a certain degree of self-hypnosis. It is probable that this phenomenon serves to explain some of the feats of the fakirs of India who apparently can render themselves insensitive or less sensitive to pain at certain times.

One factor which seems consistently to reduce reactivity to pain is muscular relaxation. Later, when we consider some of the characteristics of muscular tension and some of the behavior which it facilitates, we shall find that anxiety is one of the prominent symptoms associated with marked tension in striated muscles. Conversely, as the amount of tension in these muscles is reduced, so too is the anxiety. The same appears to be true for pain and for reactivity to other stimuli. Some work indicates that sensory thresholds are raised under conditions of extreme muscular relaxation, but these findings need verification.

Reactivity to pain also is reduced when one is under the influence of **sedatives,** that is, drugs that depress the CNS, and if sedation is carried to the point of stupor and sleep the pain threshold itself is raised. General anesthetics represent the extreme effects in this class of drugs, but many a ship's doctor will confirm that alcohol is a good substitute if no general anesthetic is available.

Factors that Increase Pain. At least two factors may be cited that lower our pain threshold: inflammation of tissue, or an inadequate blood supply in a particular part of the body. Any inflamed area, such as produced by a sunburn or an infection, will show a lower pain threshold. And, strangely enough, when blood supply is markedly reduced (that is, when **ischemia** occurs) in a given body area it will become hyperalgesic. Witness your own sensations when, as you probably describe it, your leg has "gone to sleep." You mean that you have inadvertently interfered with the blood supply; and stimuli within the leg itself, to which ordinarily you are insensitive, now become quite painful. In case your own arteries are so well protected that you have never experienced this sensation, you can demonstrate it to yourselves quite easily by applying a tourniquet to an arm just above the elbow. Movements of the fingers and wrist will soon become painful. This is called ischemic pain (cf. Table 8-1).

Referred Pain. Referred pain means that a pain which originates in one place is felt to occur at another particular place, usually on the body surface. It is common knowledge that in the heart condition known as angina pectoris the pain is referred to the shoulder and underside of the arm. And low back pain may be referred from a number of different organs, including colon and uterus, making diagnosis difficult for physicians. The pain typically is referred to that area of the skin whose nerves enter the spinal cord at the same place as those from the visceral organ in question. The simplest explanation of referred pain is that the visceral pain fibers end upon the same secondary neurons in the spinal cord as do the cutaneous pain fibers. Localization, therefore, is confused and the cutaneous reference usually is dominant, probably because we have had more experience with cutaneous than with visceral pain.[4, 5]

Reaction to Pain. To explain many complex and puzzling phenomena arising in the study of pain, it has been useful to make a distinction between the sensation of pain and the reaction to pain. Even casual observation of reactions to having a tooth filled indicates that some people take it as a matter of course whereas others make a fuss about it. A study which compared the responses of normal and neurotic individuals to pain in an experimental situation showed that, although their pain thresholds were similar, the neurotics reacted to it more and would not bear as much of it.[6] The reverse phenomenon occurs after an operation called ***prefrontal lobotomy.*** This is an operation in which nerve fibers leading to and from the prefrontal lobes are severed from the rest of the brain and which is performed sometimes for the relief of unbearable pain. When questioned after such an operation, the patient often admits that his pain is still there, but that it no longer bothers him; and he often is able to carry on daily activities of which he was incapable before the operation. The sensation of pain and the reaction to it are, thus, not the same and reaction cannot be used as an indicator of amount of pain. We must depend upon psychophysical measurements for the description of pain.

Pain, Feeling, and Emotion. Is pain the opposite of pleasure? Is pain an emotion? Is pain a feeling? In the first place, pain is primarily sensation. We interpret such a sensation; that is, we perceive pain — and the perception itself is the " feeling." But over and above this perceptual aspect of pain is another quality of experience. We often say, during the perception of severe pain, that we are excited, we are emotional, or we are overwrought. What do we mean by these words? Actually, as we shall find in our study of emotion, there is not much agreement about what we do mean. This much is certainly true, however, pain arouses the autonomic nervous system. When we perceive intense pain we turn pale, we perspire freely, and many internal changes take place, all of which are autonomic responses. The autonomic nervous system influences responses of smooth muscles and glands. Some of these responses set up nerve impulses which are in turn carried to the cortex and are again perceived, sometimes as secondary pain, more often as drawing, pulling, lumps in the stomach, feelings of swelling, or other feelings of distress.

A more detailed consideration of what this means will occur in the chapter on emotion, where we shall find that pain may have distinct emotional components in addition to its perceptual character; and we shall find that it is different from, but not necessarily " the opposite " of, pleasure.

Suggested Readings

1. Morgan, C. T., and Stellar, E., *Physiological Psychology*, 2nd Ed. New York: McGraw-Hill, 1950, pp. 230–273.
2. Jenkins, W. L., *Somesthesis*, S. S. Stevens (Ed.). *Handbook of Physiological Psychology*. New York: Wiley, 1951, Chap. 30, pp. 1172–1190.
3. Ruch, T. C., " Somatic Sensation " (Chap. 16), " Neural Basis of Somatic Sensation " (Chap. 17), and " Visceral Sensation and Referred Pain " (Chap. 19) J. F. Fulton (Ed.). *A Textbook of Physiology*, Philadelphia: Saunders, 1949, pp. 292–315, 316–347, 360–374.
4. Edwards, W., " Recent research on pain perception," *Psychological Bulletin*, 1950, 47, 449–474.

9 | *The Proprioceptive Senses*

This chapter is concerned with **proprioception.** If you will refer to Table 7-1, you will see that proprioceptors are an important class of receptors. They are those sense organs located in muscles, tendons, and joints, and in the nonauditory labyrinth or inner ear; they respond largely to bodily movement. The muscle, tendon, and joint senses are collectively called **kinesthesis.** They are related because they are normally stimulated together and cooperate closely in the control of movement. The senses which depend upon receptors in the nonauditory inner ear (the *vestibule*) are sometimes called the labyrinthine senses. We shall refer to them collectively as the **vestibular sense.** Here, also, one finds a number of different receptors with different characteristics of response. They are all stimulated by head movements of various kinds and give rise to a number of important reflexes. They are of great importance in both static and dynamic balance.

It is difficult to illustrate the importance of proprioception without resort to gross abnormalities or to animal experimentation, because proprioception is in a large measure "silent," at least as far as adult awareness is concerned. Hold your arm out straight at shoulder height and move the forearm back and forth several times, attending to the "feel." There is not a great deal of information, psychologically speaking, and the most prominent sensations are in the elbow joint and in the pinched or stretched skin, but you know where the arm is and what it is doing. Whoever doubts the importance of these sensations has but to watch a person with tabes dorsalis (destruction of the kinesthetic neurons of the spinal nerves by tertiary syphilis) trying to walk. He can walk only by watching his feet because he has no other way of knowing where they are. The walk is very poorly coordinated and the feet slap — it bears little resemblance to the normal, springy gait of one whose kinesthesis is intact. It is not only in the control of movement that kinesthesis is involved but also in the sense of position. Knowledge of position is part of the information which the nervous system must have to initiate accurate, appropriate movements because the direction, force, and speed of movement depend partly upon where the limb was at the beginning of the movement. In addition, kinesthesis is involved in many reflexes having to do with posture, walking, balance, and coordination, and in the learning of motor skills.

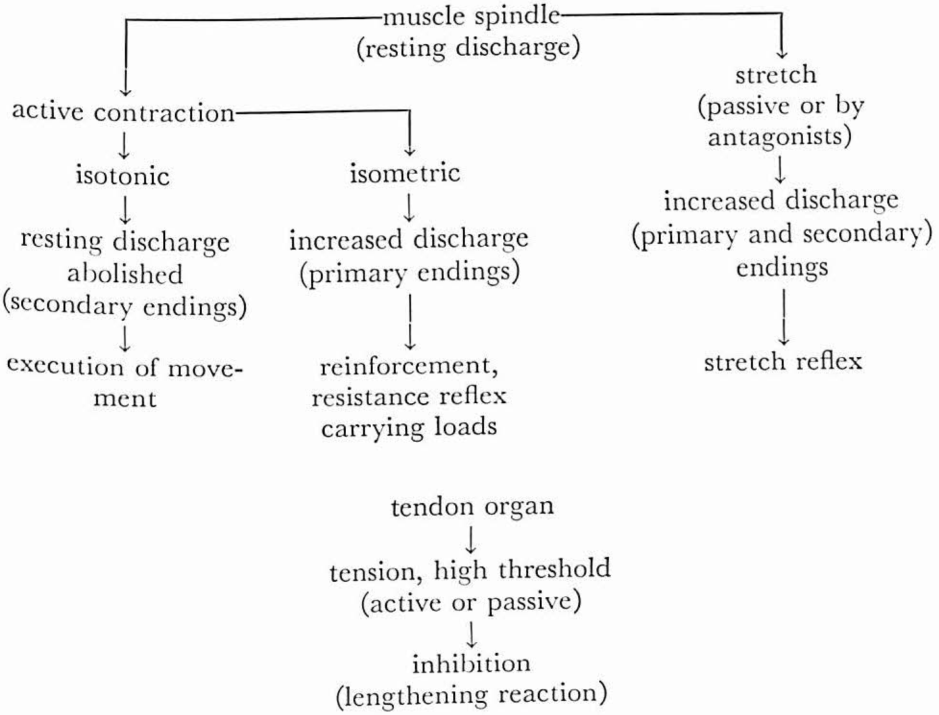
The vestibular sense is involved in reflex behavior which is basic to maintenance of muscle tonus, to proper adjustments to vigorous movements that tend to throw the body off balance, and to displacement or rotation of the head. Maintenance of muscle tonus is necessary for prompt and adequate response — a flabby muscle is not ready to respond. A vigorous movement tends to throw the body off balance and preparation for meeting this emergency is in part the duty of the vestibular senses. Turning the head sharply, jumping, or otherwise subjecting the head to rapid accelerations sets up reflex adjustments through the vestibular mechanisms. In normal life, these reflex adjustments stand us in good stead, but at times they cause us a good deal of distress. Seasickness is largely a result of stimulation of the vestibular mechanisms and it can be crippling temporarily. A disorder of the inner ear known as Meunière's disease can cause such extreme nausea and dizziness as to render the patient helpless. Finally, vestibular reflexes can cause the pilot flying blind a vast amount of trouble and he must learn to disregard them and trust his instruments.

Kinesthesis

Kinesthesis may be defined as that group of senses involved in the perception of movement and position, and whose receptors are located in the muscles, tendons, and joints. There are at least four different kinds of receptors involved in these senses and the stimulus for each is somewhat different. It is not, therefore, profitable to attempt to discuss stimuli apart from the discussion of the receptors and their central nervous connections.

Within the muscle are found specialized fibers which do not contribute to the actual contraction of the muscle, but carry the two types of **muscle spindle** receptors. The so-called primary afferent fiber from the muscle spindle is the large fiber connected to the **annulo-spiral ending.** The so-called secondary afferent, smaller fiber from the muscle spindle innervates the **flower-spray ending.** Both of these may be seen in Figure 9.1. It is not the contraction of the muscle but its stretching which is the stimulus for these kinesthetic receptors.

Table 9-1. Summary of Mammalian Proprioceptive System
[After Lissman, (1950)]


muscle spindle
(resting discharge)
active contraction
isotonic
resting discharge abolished (secondary endings)
execution of movement
isometric
increased discharge (primary endings)
reinforcement, resistance reflex carrying loads
stretch (passive or by antagonists)
increased discharge (primary and secondary) endings
stretch reflex
tendon organ
tension, high threshold (active or passive)
inhibition (lengthening reaction)

Both kinds of muscle spindle receptors show a resting discharge; that is, nerve impulses of low frequency occur in the afferent nerves when the muscle is at rest. This resting discharge is modified by what happens to the muscle — it increases in rate if the muscle is stretched, it may cease completely if the muscle contracts. These receptors are responsible for the stretch reflex (cf. Chap. 16).

A different kind of ending is found in the tendon. Figure 9.1 shows the location of the tendon organ. It is embedded in a bundle within the tendon and is innervated by a large-diameter fiber. These receptors are stimulated by tension and they, therefore, respond when the muscle contracts, putting tension upon the attached tendon. The threshold for these receptors is rather high, about 100 times that of the annulo spiral endings. The tendon organs are assumed to be responsible for the lengthening reaction found in decerebrate rigidity (cf. Chap. 16). In any stretch or contraction of a muscle a particular pattern of proprioceptive impulses will arise, depending upon the relative activity in each of the three kinds of receptors involved. It is the pattern upon which we depend for our kinesthetic cues. Table 9-1 shows some of these relationships.

The fourth type of kinesthetic receptor is the Pacinian corpuscle. which is found abundantly in and near the joints. It may be seen in Figure 7.1 in the deeper tissues under the skin. These receptors respond to pressure upon them, such as occurs when a joint moves. Little is known about joint sensitivity, but it is probable that other receptors are also involved. Pacinian corpuscles, in common with other kinesthetic receptors, show a resting discharge and little evidence of adaptation with moderate stimuli over long periods of time. In this respect the Pacinian corpuscles, although responding to pressure, are unlike the touch receptors.

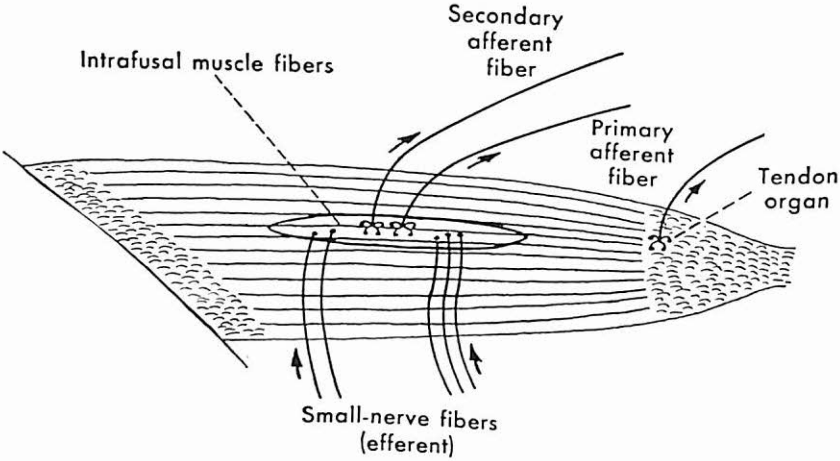


FIG. 9.1. Schematic diagram showing innervation of a muscle spindle and a tendon organ. (Redrawn from Kuffler and Hunt.[23])

The central nervous connections of the kinesthetic receptors are poorly understood. There are several lines of evidence which indicate that there are a number of different possible pathways, levels of crossing in the spinal cord, and connections at higher levels. It is possible, but not definitely known, that different functions within the total kinesthetic system travel by different routes and recent evidence indicates that the spinal cord must be almost transected to eliminate them completely.[1] To confuse matters further, there is some suggestion that the stretch reflexes (knee jerk, for example) are eliminated by a .2-percent procaine solution in the spinal fluid, whereas " position sense " is unimpaired.[2]

In the present state of our knowledge about the diverse kinesthetic senses, it is difficult to make definite statements about cortical localization. The evidence is fairly good that the receptors in the muscle spindle, responding only to stretch, do not give rise to cortical potentials but give localized responses in the cerebellum.[3] This would indicate that perhaps the function of these receptors is entirely reflexive and their activity does not give rise to awareness. There are other lines of evidence, psychological in nature, which lead to the same conclusion. Other kinesthetic receptors, however, appear to give rise to potentials in the contralateral cerebral cortex. The area involved (the post-central gyrus) is the same as that for touch. The impulses arise from receptors in tendons and joints, and these are the senses which yield better awareness of position and movement than the muscle senses.

Qualities in kinesthesis are indescribable. There is apparently no " feel " at all from the muscle senses. The tendon " feel " is that pull that occurs upon stretching the arm out as far as possible. The joint " feel " is best attended to by resting the relaxed hand on a table and passively moving a finger with the other hand, slowly and gently. One does feel soreness in muscles after unaccustomed exercise, but the origin of these sensations is not known. Since they are painful, we would assume that free nerve endings of the pain sense are in some way stimulated, possibly chemically in this instance. In connection with kinesthetic stimulation, one also has accompanying sensations of cutaneous and deep pressure which are unavoidable, except in animal experimentation, where these are first eliminated. Thus, it is not possible to obtain threshold measurements for each kind of sensitivity; in all such measurements the threshold is one for the activation of several or all senses involved. The patterns of activation will vary considerably from one kind of task to another, so that the results obtained are dependent upon the particular muscular task and may not hold for a different one.

One of the most frequently used procedures for the investigation of kinesthesis, as well as one of the earliest for the investigation of psychophysical problems, is that of lifted weights. In this kind of study, carefully graded weights of identical size and shape are lifted by the subject with the same motion and in the same fingers, according to experimental plan. Differences of perceived weight are due largely to differences in kinesthesis and the DL's so obtained are taken as kinesthetic DL's. The DL increases from about 3 grams at a standard of 25 grams to about 50 grams at 600, showing fair approximation to Weber's law.[4] The DL depends also upon the part of the arm used for the lifting motion. It is higher for the wrist than for the elbow and least for the shoulder. The shoulder also has been shown to be a very sensitive joint in threshold to both passive and active movement. The absolute threshold for passive movement, at a rate of .3° per second is about .3°. Other joints investigated have not been as sensitive.[5]

Both these experiments indicate that it is the joint which is affording most of the information upon which kinesthetic judgments are made. Further evidence comes from clinical studies where loss of joint sensitivity was accompanied by great reduction in kinesthetic cues, whereas loss of sensitivity in skin and muscle was not. These conclusions strengthen those based upon the physiological evidence, previously presented, that joint and tendon sensitivities are more important psychologically than are muscle sensitivities. This in itself is a surprising conclusion, but there are more surprises in the field of kinesthesis which we shall find in due course.

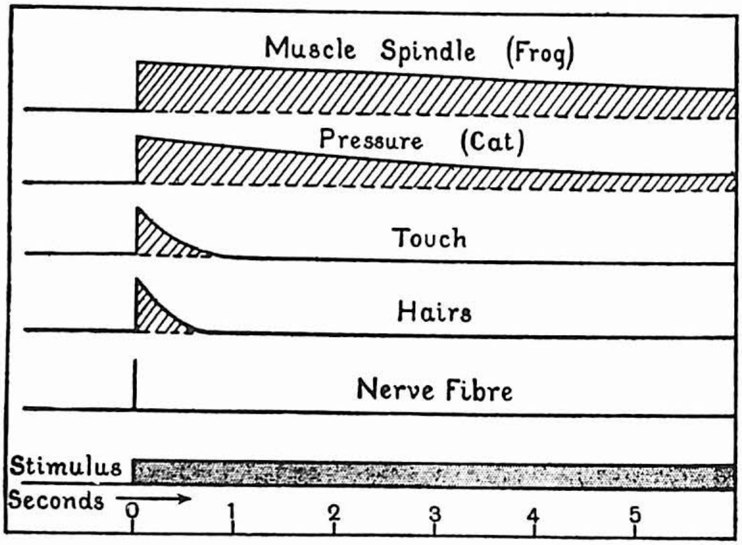


FIG. 9.2. Diagram showing rate of adaptation to a continuing stimulus. Adaptation is slowest in the muscle spindle. Note the single brief response of the nerve fiber without receptor. (After Adrian.[24])

Just as qualities are peculiar in kinesthesis, so is *extent*. The category is not obvious to introspection. It may be because whole areas are usually involved in any stimulation; the variation of extent is small and localization is correspondingly poor. *Duration* is another matter, but here again kinesthesis is different from other senses. Kinesthetic sensations, if mild, endure for long periods of time, with relatively little change.

Figure 9.2 shows diagrammatically the relative rate of *adaptation* in several kinds of receptors. The top diagram shows the rate of adaptation for the muscle spindle receptors; it shows little change. Other evidence indicates that, if the stimulation is not intense, very little adaptation occurs in any of the kinesthetic receptors. Afferent nerve fibers from these receptors show a resting discharge which is then modified by stimulation. This is coupled with unusual resistance to adaptation on the part of the receptors. It would be rather awkward if adaptation occurred rapidly in antigravity muscles, for we would often find ourselves sitting down in the most unexpected and unwelcome places! And certainly our precision of control of movements would be far poorer after an hour or so at a typewriter or on the golf course. An attempt to assess kinesthetic adaptation was made by means of the lifted weights technique. It was found that both time and intensity were factors in adaptation. Figure 9.3(a) and (b) show the results in terms of the time needed to recover from various exposures to known kinesthetic stimuli. The longer a weight is held, or the heavier the weight that is held prior to the determination of the DL, the larger the difference between weights must be to be perceptible to the subject. He has, in other words, become less sensitive; that is to say, he has adapted.

FIG. 9.3A. Kinesthetic adaptation: the recovery time as a function of adapting the intensity; standard time was 1 minute. (After Holway, Golding, and Ziegler.[25])

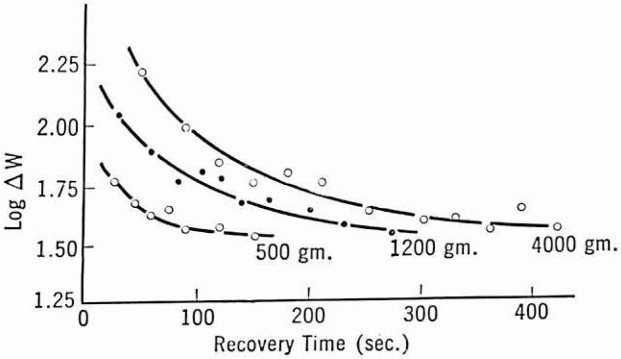
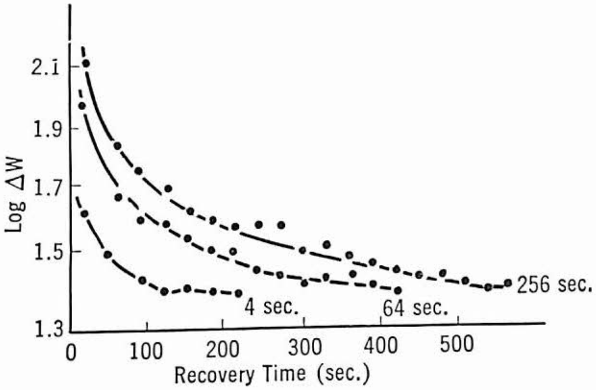


FIG. 9.3B. Kinesthetic adaptation: the recovery time as a function of adapting the duration; standard weight was 4000 gms. (After Holway and Ziegler.[26])



After-effects are not prominent in kinesthesis, but they can be shown to exist. Stand in a doorway and press your arms against the two sides rather strongly for 30 seconds or so. Then step out into the room and relax. Your arms will rise most gracefully as an aftereffect of the previous stimulation.

The Role of Kinesthesis in Posture and Movement. Consideration of the tremendous number of muscles and of muscle fibers cooperating in any maintenance of posture and in any movement, no matter how apparently simple, and of the nicety of cooperation among them to produce a movement which is smooth, well-timed, and of proper direction and force, leads one to suspect that the kinesthetic senses actually do a great deal of work, even though they may not play a large part in perceptual awareness. The kinesthetic system is not a simple one, involving as it does many reflexes and central nervous system structures. Even the maintenance of a normal resting, relaxed posture is not uninfluenced by kinesthetic recircuits, for tonus is maintained in muscles and any slight movement starts from an appropriate base. Studies in the control of movement have led to some extremely interesting results. That kinesthetic distance or space is different from visual space is not always appreciated. Judgment of distance by kinesthetic means makes the distance seem much longer than a visual distance; using a 1-inch visual standard, a kinesthetic dsitance seems almost 50 percent longer.[6] Judgment of kinesthetic distance is influenced by the load which is carried — the heavier the load, the shorter the distance which is traversed to match an unloaded, standard kinesthetic distance.[7] It has also been found that kinesthetic judgment under load makes an area seem larger, makes an angle traversed seem more acute, makes an arc seem more sharply curved.[8]

That kinesthesis is important in the control of movement is unquestioned, but the exact role which it plays is not always obvious. Studies of kinesthetic reaction time — reaction time to the passive dropping of the arm — have reported reaction times of 120 to 130 milliseconds. These are relatively rapid as far as simple reaction times go (auditory reaction time is about 150 milliseconds) but they are not fast enough to permit voluntary control of movement through kinesthetic feed-back circuits. The maximum rate of reciprocal movements, as in tapping or speech, appears to be about 8 to 10 per second, the contractions lasting about 50 milliseconds. Since tapping at a rate of 8 per second really requires 16 changes of hand motion, the kinesthetic reaction time, if it were to serve to control the movements, would have to be no more than about 60 milliseconds, twice as rapid as it in fact is.[9] The feed-back analogy, at present a popular way of explaining control of movements, thus will not explain it. It has been suggested that the control is exercised by the higher centers which merely select the desired response pattern and send the proper impulses to the lower centers involved, which then execute the movement. Any correction of a movement once under way is difficult, if not impossible. That there is some truth in this contention is evident in the difficulty with which a batter halts or changes his swing, once he has made up his mind to swing at a ball, even if the pitch is wide of the mark. And it is equally difficult even to check a golf swing, once started. One can spoil it sometimes, but rarely improve it; the golfer is usually aware, from the moment he starts his down swing, of whether the ball will be well hit or not.

What then does kinesthesis do in the control of movements? It is undoubtedly important in dealing with and correcting slow movements. It is also necessary in the learning of skillful movements. Probably the most important thing it does is to give prior information about the limb's starting position, the existing state of tonus, and, during the course of the movement, whether it was smooth or awkward and of improper force. The last of these could conceivably be used in learning by requiring the trial and error correction of the command pattern sent down from higher centers. It has been further shown that sequential stimulation, as in tracking problems (used in gunnery), does not evoke continuous, smooth response. To make a second movement after a first is under way requires a lag of .5 seconds.[10] The rate at which corrective movements are made in a tracking task suggests that they must be ballistic movements dependent upon the initial impulses for accuracy. Three investigators have reported that rapid movements cannot be corrected as they are in progress and, thus, stress the importance of the cortical organization of movement.[11, 12, 13] Recent studies also have shown that tracking can be greatly aided by improving the visual information given by the system itself,[14] so that some learning of skills may be largely a learning to respond to minimal cues. Any means which can be found to make the cues more obvious could be expected to result in much faster learning. The learning of motor skills is not entirely a kinesthetic problem but involves total perceptual organization.

Of some practical importance are studies of the accuracy of reaching for controls. In general, reaching is more accurate directly in front of one and accuracy decreases as the distance from the starting point increases. Individual differences are large, but accuracy and speed are very highly correlated (a common finding in many psychological functions). Average accuracy in one experiment was 3 inches.[15] These findings are of importance in the design of aircraft controls, the reason for the original experiments, but also have application in designing automobile controls and controls for industrial machinery.

Kinesthesis plays a role in the maintenance of posture, as already indicated. One such function is the providing of information of where each part of the body is, and what it is doing, as a basis for the maintenance of posture under diverse conditions of movement and position. Many of the important postural mechanisms are reflexive in nature and it is important for the physiological psychologist to understand them because they are the underlying substrate without which normal movement, posture, and behavior is impossible. Although later discussion is essential to a real understanding of the significance of reflexes (cf. Chaps. 16, 18, and 19), it is necessary to mention the reflexes which are kinesthetically initiated, because these are the main functions of kinesthesis. Most of the work on reflex behavior has been performed on four-legged animals (mostly cats and dogs) and may appear to have little direct bearing on human beings. Actually, however, these fundamental mammalian reflexes are still of importance in behavior and underlie our own behavior patterns. Because of the encephalization which has proceeded apace, studies of human beings would be of doubtful value in a study of reflex mechanisms. The fact that we stand on two legs instead of four, and do not have very good righting reflexes if dropped from a height, does not mean that a study of these will not throw light on how we maintain balance or recover balance if tripped. An important principle of reflexive behavior is reciprocal innervation, which was first introduced as a concept in Chapter 5. Reciprocal innervation is kinesthetically controlled and refers to reflex relaxation induced in the antagonist as the agonist contracts. Without some such provision the organism could readily get into a state of complete immobility because of simultaneous contraction of all the muscles of a limb. Reciprocal innervation is important in the stepping response and is thus necessary to walking.

A kinesthetically controlled reflex which is necessary for the maintenance of posture is the stretch reflex (cf. Chap. 16). The stimulus is the stretch of the muscle itself (not the tendon). The reflex response, the development of tension in the muscle which has been stretched, is the basic postural reflex, resisting the gravitational pull. The crossed extension reflexes are also important. Kinesthetic stimuli in a limb on one side of the body give rise to reflex extension of the limb on the opposite side and, also, of the hind limb on the same side, thus maintaining a nice balance against gravity. The animal could not, of course, flex one forelimb without making some adjustments in other limbs, for he would be thrown off balance and would fall. In your own walk, although you are balanced on your hind limbs, notice the motion of your arms — they swing so as to balance your body; any other motion is exceedingly awkward.

There are other important kinesthetic reflexes, the tonic neck reflexes, which arise from the neck muscles when the head is twisted and turned in various ways. It should be noted that these neck reflexes are so called because they are initiated by the receptors in the neck muscles, but that the effects are felt all over the body; the balance of the entire body is involved. These reflexes cooperate closely with the vestibular reflexes and with the other reflexes involved in posture and movement. The whole complex cooperative mechanism is so nicely integrated and functions so smoothly that we are seldom aware of the fact that anything very amazing is occurring. It takes but a moment's reflection on how different our behavior would be if we had to devote our attention and efforts to taking care of these " minor details " to bring a realization that reflexes are indeed important topics for study.

A word about some of the less obvious uses of kinesthesis may be of interest. The first of these is the development of depth perception, where kinesthesis plays two roles. First, it is of some importance in providing cues of accommodation and convergence, which act, within very limited distances, as binocular cues to depth. The second way in which it is influential in depth perception is in the learning of depth and distance perception itself. Good judgment of depth and distance is not present at birth, not, at least, in the human being and closely related species. There is also no reason to suppose that it comes about through maturational processes. We learn to see depth because we have had a great deal of experience with seeing things and then walking or jumping to them. That such judgments must be reasonably accurate is easily demonstrated. You would, if there were some reason for it, jump from a 4-foot wall, for you can prepare your muscles so that it will do you no harm. You would not (if you are an average person) jump from a 20-foot wall. But you must be able to tell the difference! It is not intended to imply here that kinesthesis is the only factor in the development of depth perception, but only that it is an important one which is often overlooked.

Another rather neglected aspect of kinesthesis is the role it plays in the development and maintenance of the body image. One can perceive only when one has sense organs to underlie the process — we do not have immediate intuition of objects, even if that object be ourselves. We know only those things about ourselves for which we have senses to give us the information. Among the important ones are the kinesthetic senses. They inform us where our limbs are, what they are doing, how tense or relaxed we are, what position we are in, how smoothly we are performing some skilled act, how much effort some act takes, whether we are fresh or tired, and may even contribute to such feelings as optimism or depression, euphoria or lethargy. These sensations have always been with us, as a backdrop against which the play of life goes on, since birth or before. They are part of our memories, our images, our ideas of how to do things or of how well we do things. They are part of the "self-object," part of what it means to be you, and no one else. They are basic to the feeling that your body belongs to you and it is impossible to imagine yourself without them — so impossible that most people never consider the problem at all.

A final area in which kinesthesis is important is in the study of rhythm. Rhythm is important in two quite different areas, in efficiency of movements and in esthetics. Natural rhythms for bodily movements can be utilized to advantage in performing work with least expenditure of effort — walking in the preferred rhythm is less tiring. A full discussion of the problem would take us too far afield, but it may be said that it is in part rooted in kinesthesis. Rhythm is probably the most basic esthetic expression in the entire animal world. Not only do most human beings, of widely scattered cultures, produce rhythms of many kinds for pleasure, but a kind of rhythmic motion, closely resembling primitive dancing, can be observed in the anthropoid apes. An appreciation of rhythm underlies all dancing and all music, although it receives varying emphases, depending upon other elements present. All people seem to enjoy rhythm; children often indulge in rhythmic movements similar to dancing, purely spontaneously. It is likely that one of the elements in sports which make them so popular is this same basic delight in rhythmic movement, which can no longer be indulged in, in the purely spontaneous way open to children, for society frowns on such levity.

THE VESTIBULAR SENSE

The labyrinth or inner ear is a peculiar and complex structure embedded in the temporal bone of the skull at approximately the level of the *pinna*, which you probably call your ear. It is divided into two major parts, called auditory and nonauditory, the first having to do with hearing, which we shall discuss later, and the second having to do with posture and balance, which we shall be concerned with here. The labyrinth is so named because it is a tortuous, maze-like cavity in the skull. It is lined with membranous canals, which are in turn filled with a viscous liquid, the **endolymph.** All parts of the labyrinth intercommunicate through small openings. Figure 9.4 shows the relationships among the parts.

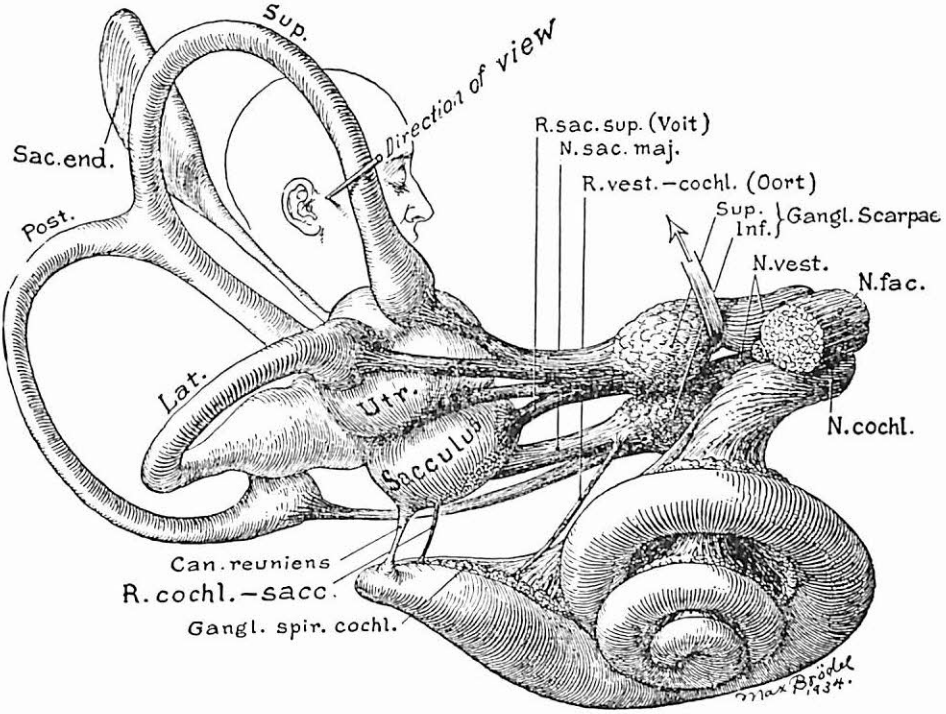


FIG. 9.4. Diagram of the inner ear, showing the semicircular canals, utricle, saccule, and cochlea, together with the nerves innervating them. (After Hardy.[2])

The nonauditory labyrinth, or vestibule, is again divided into several parts. They are shown schematically in Figure 9.4. The most prominent are the **semicircular canals,** three looped, semicircular structures, lying in three different planes of the head. The anterior and posterior canals are in vertical planes, about at right angles to one another and at about 45° from a plane parallel to the midline of the head. The third canal is the horizontal canal. There are canals on both sides of the head and they are so placed that the anterior canal of one side is parallel to the posterior canal of the other side. The other parts of the labyrinth are called the **saccule** and **utricle.** The semicircular canals connect to the utricle, which in turn is connected to the saccule (Fig. 9.4).

The stimuli for each of these three organs are best considered along with the discussion of the actual receptor mechanisms but, in general, it may be said that all of them are stimulated by acceleration of the head — linear, angular, or both.

At one end of each semicircular canal a small swelling, called the **ampulla,** may be seen. Within the ampulla there is a mass of receptors cells, collectively called the **crista,** from which fine hair-like structures extend into a gelatinous mass, the **cupula.** The cupula completely fills the cavity to the opposite wall, so that there is very little leakage of endolymph as the cupula slides back and forth over the hairs of the crista in response to rapid turning of the head. These structures may be seen in Figure 9.5A. The adequate stimulus for these receptors is angular acceleration, such as would occur in rapid twirling or rotation. No matter what the plane of rotation of the head, one or more of the canals is in a position to be strongly stimulated by the acceleration; the canal lying in the plane nearest the plane of rotation is always the most strongly stimulated. There is also some evidence that linear acceleration, and possibly position, are to some extent stimulating. Inadequate stimulation of these organs is easy by means of hot or cold water circulating in the **external auditory meatus.** Many people have experienced this during the removal of wax accumulations from the ear. The hot water circulated in the external canal makes one very dizzy and it is often difficult to walk in a straight line for a few moments. The dizziness which children enjoy, produced by whirling round and round, is also a result of stimulation of the semicircular canals, but, in this case, adequate stimulation.

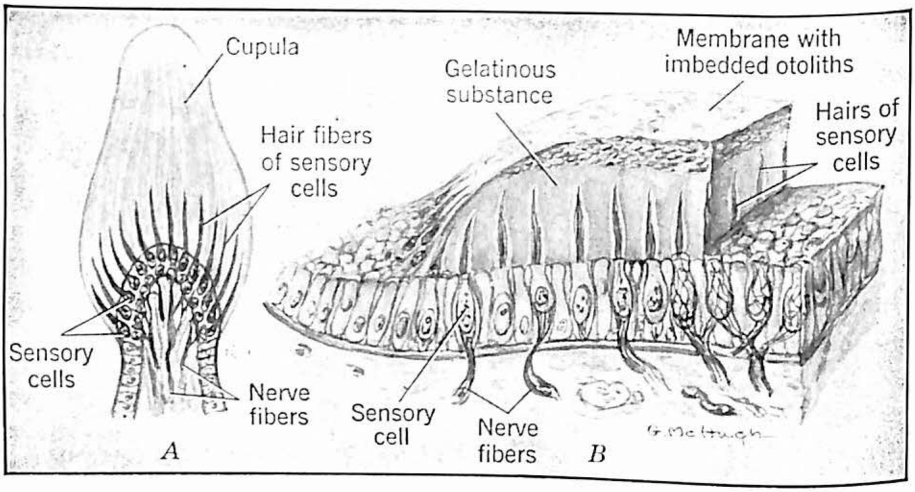


FIG. 9.5. (*Left*) crista and cupula. (*Right*) diagram of macula. (After Geldard.[28])

The receptors in the saccule and utricle are in some ways similar to those in the semicircular canals. The receptors proper are hair cells which lie within the *maculae* shown in Figure 9.5B. The *maculae* also contain a gelatinous mass and small calcified particles, called otoliths, which act on the hair cells under the influence of some force and, thus, stimulate them. The utricle is stimulated both by the action of gravitational forces and by linear acceleration. The saccule is not as well understood, but appears to have similar functions and to respond to both gravity and linear acceleration. There is some evidence that it also may serve as either a low-tone auditory receptor or as a vibratory receptor or both. There appear to be greater complexities in these structures than has been previously believed. There may be a number of kinds of receptors for different kinds of positions and movements, but their significance for understanding human behavior is not clear.

The CNS connections of the vestibular systems are both complex and unusual. All the receptors are innervated by branches of cranial nerve VIII, the acoustic nerve, these branches being called collectively the vestibular branch. The vestibular nerve sends one branch to each semicircular canal, one to the utricle and, it appears, two to the saccule (possibly only one of these serves proprioceptive functions). The afferent fibers run in these nerves to the various vestibular nuclei in the medulla, where they synapse. Connections from the vestibular nuclei are mainly to the cerebellum, to the spinal cord, and to the midbrain. Since the cerebellum is largely concerned with the smoothing, control, and regulation of force of movement, the business that the vestibular systems have with it is obvious. The vestibular nuclei have direct connection with movement systems, in this case the final common path. The higher connections are largely with the cranial nerve nuclei of nerves III, IV, and VI — those concerned with eye movements — and some cells at the level of the red nucleus in the midbrain, which is also concerned with the regulation of movement. We shall see soon what direct effects upon eye movements stimulation of the vestibular system produces.

It is interesting to note that anatomical connections to the thalamus and cortex have never been described. It will be recalled that sensation has been attributed to excitation of the appropriate area of the cortex; what, then, of vestibular stimulation? As a matter of fact, the vestibular " sense " apparently has no sensations of its own; no sensations peculiar to stimulation of any of these receptors have been agreed upon. One does, it is true, experience sensations when the receptors have been activated, but the sensations are those arising from other receptors which have been subsequently or concurrently stimulated — the skin stretched, the muscles pulled, the deeper receptors in ordinary tissues stimulated by the reflex effects induced by vestibular stimulation or by the motion itself. It would thus be reasonable to conclude that there are no sensations because there are no known cortical projections for this system, but there is some recent evidence which indicates that this may not be quite true. Cortical potentials have been recorded from the brain of the cat upon stimulation of the vestibular nerve or the vestibular receptors.[16] They appear in a small area near the auditory and, also, near the somesthetic area and are very susceptible to anesthesia. Anatomically no connections have been traced. If this physiological evidence is confirmed, then some modification of our preceding statements will be necessary. In any event, the cortical representation is not the salient thing about the vestibular system and the subcortical connections are of major importance.

Records of action potentials from the vestibular nerve show resting potentials, as in other proprioceptive systems. It is on this basis that the semicircular canals (the records in this case taken from the branches serving the ampullae) are considered to be static as well as dynamic receptors. This basic frequency may be modulated in either direction. Ordinarily it is increased by ampulla-trailing rotations and decreased by ampulla-leading rotations. Deceleration produces the opposite effects, as one would expect from the other experimental data. The otolith organs show similar constant activity, but the modification of pattern is more complex.

Effects of Stimulation. As we have seen, there is no unique sensory quality associated with the arousal of the vestibular system. It follows that there is also no extent, no duration, and no intensity. Thresholds are thresholds for arousal of reflex effects of various kinds or for perception of movement and are not true sensory thresholds. Before proceeding to a discussion of these thresholds, we must first consider the more prominent reflex effects of vestibular stimulation.

One of the most prominent effects of vestibular stimulation, from the point of view of the observer watching a subject in a rotation experiment, is a peculiar series of eye movements, called **nystagmus**. In nystagmus, the eyes drift slowly in one direction, then jerk rapidly back to an approximately central position, whence the slow drift starts again. This sequence of movements may be either the result of stimulation or of the after-effect of stimulation, the direction of the slow drift being opposite in the two cases. It is called primary nystagmus if it is the result of stimulation. Since most situations for experimental stimulation of the vestibule utilize the rotation chair, the effects of stimulation are inextricably mixed with the after-effects of this stimulation and secondary nystagmus is imposed upon the total pattern. Secondary nystagmus is the inverse of primary nystagmus. If the rotation is in the clockwise direction, i.e., to the right, then the rapid movement is to the right, the slow drift to the left. The after-effect is in the opposite direction. If the head is placed in unusual positions, various effects are obtained, depending upon the canals most strongly stimulated.

The tonic labyrinthine reflexes are important sources of proprioceptive control. They cooperate with the tonic neck and other reflexes in a pattern which is of great importance in lower mammals, the righting response. If a cat is dropped from a height of several feet, after being held with its back to the floor, it will, nevertheless, land on its feet; not only on its feet, but with the proper tonus in appropriate muscles so that it can support the weight of the body when it lands. The first phase of this righting pattern is elicited by the stimulation of the labyrinths and if the labyrinths are destroyed the animal cannot right itself. Once the head has been turned, in response to the vestibular stimuli, then the neck reflexes (kinesthetic) are called into play. The vestibular stimuli also cause the forelegs to be extended, so that greater weight can be borne without falling, and the toes to spread to give a broader base and more stable balance. Although these reflexes are difficult to demonstrate in the human adult, they are present in the human infant and undoubtedly underlie much of the postural and movement behavior in the adult. The preparation which is made for landing from a jump, the extra tonus in extensor muscles to take the added weight, the spacing of the feet apart, the spreading of the arms to afford better balance, are all to some degree initiated by the vestibular stimulation attendant upon acceleration of the body (and therefore the head).

There are other tonic and postural reflexes which are the result of vestibular stimulation. There is a general tonic effect upon the extensor muscles in general; extensor tonus is lost if the vestibules are destroyed, as by disease or operation. There are also effects elicited by position alone, not by movement or acceleration. In experimental animals, the supine position, with the snout elevated at about 45°, results in maximal extension of the limbs. The position for minimum extension of the limbs is just 180° different. In between these two positions occur intermediate amounts of extensor tonus. This, then, is a purely positional reflex and is presumed to be dependent upon the utricle.

Rectilinear movement, particularly movement up or down, which does not throw the body off balance, gives rise to reflex adjustments of the legs. These can be observed in an elevator, especially if the accelerations are rapid. Upon rapid acceleration downward, the legs extend. The reverse occurs if the acceleration is upward. The stimulation which occurs when rotation to which one has become adapted is suddenly stopped gives rise to adjustments of the entire body. If the rotation has been to the right and the stop is sudden, then the rapid phase of the resulting nystagmus is to the left, the head turns to the right, the trunk and arms do likewise, the arms and trunk tend to dip to the right also, and the legs make adjustments which would be appropriate if the stimulation were actually primary stimulation, that is, the left one extends and the right one flexes, in order to keep the balance. If stimulation occurs with the head held in an unusual plane, the resulting forced movements will throw the subject violently off balance, and he may do himself serious damage if not supported. The perception accompanying these contortions (so they appear to the observer) is of movement of the environment, and a strenuous attempt to keep one's balance. These effects are sometimes pronounced in flying aircraft, particularly when vision is limited or excluded, and the pilot has to learn that they are not to be trusted, as indeed they are not in any such abnormal situation as being hurtled through the air at high speed. If the head is inadvertently moved during a rapid acceleration, the effect is multiplied and the pilot is likely to lose all control. There are a few brave psychologists who are known to have tried this when in the rotation chair and are reputed to have been bed-ridden for several days.

In addition to giving us vital information about position, direction, character, and violence of movement through space, the vestibule also produces reflex effects of nausea and reverse peristalsis. Unpleasant as these are, there are genuine vestibular effects, not purely psychogenic. The topic is of widespread interest and economic importance. A later section will be devoted to it.

Thresholds. It is difficult to cite accurate threshold figures for vestibular stimulation. First, there are apparently very large individual differences, so that an average figure may not be too meaningful if a small number of subjects has been used in an experiment, which is usually the case. Secondly, there are a rather large number of receptors of different kinds which are stimulated by any motion and two experiments which appear to differ but slightly may actually result in stimulation of different receptors with different degrees of intensities. Thirdly, there are concomitant sensations aroused which are not of vestibular origin, for which the thresholds are apparently lower than are those of the various vestibular receptors. Cutaneous touch, deep touch, and kinesthesis are aroused by the pulling of tissues when the body is accelerated. These sensations are most noticeable at the points of contact with the chair or other bodily support. Sometimes the pulling of the abdominal viscera against the mesenteries, or the stretching of skeletal muscles gives cues to bodily motion. These factors make any actual figures cited of value for order of magnitude only, rather than for precise thresholds.

The threshold itself, in any given experiment, depends upon the indicator of vestibular activity which is chosen. Many indicators are possible. Among the most popular are perception of motion, appearance of detectable nystagmus, and the **oculogyral illusion** (apparent movement of a faint light). Thresholds for rotation, involving primarily the semicircular canals, are from .2° to 5° per second^2. Using a " human centrifuge " as the rotation apparatus and the oculogyral response as the indicator of stimulation, thresholds of the order of .12° per second^2 have been found for both positive and negative acceleration, if the exposure period is brief.[17] Thresholds for rectilinear acceleration, stimulating largely the saccule and utricle, are more difficult to obtain. One study, using a see-saw apparatus, reported thresholds of 1.7 to 4.0 centimeters per second.[18]

After-effects. After-effects of stimulation of vestibular receptors are puzzling. We have already suggested that some of these after-effects are so prominent as to interfere with study of the primary effects of stimulation. If a subject is rotated to the right, the primary response is followed by an opposite response in which the nystagmus is reversed and the perception of motion is also reversed. These effects are, of course, powerfully influenced by the presence of visual cues, so that the subject's eyes must be closed in order to study pure vestibular responses. These so-called " after-images " are now thought to be the result of real stimulation, in which the cupula, in returning to position after the bend given it by the initial acceleration, overshoots and bends the other way before returning to the resting position. After the initial acceleration, rotation at a constant speed does not stimulate the semicircular canals; with the eyes closed, the subject will state that he is not moving. Stopping the rotation rapidly again induces vestibular stimulation by virtue of the negative acceleration, which bends the cupula in just the same manner that positive acceleration did at the start of the experiment, but opposite in direction. In this case, the primary effects and the secondary, or after-effects, are the opposite of what they were at the start. Rectilinear motion has been very poorly studied; it is reported that the after-effects are negative, but little is known of them. It would be expected, from the construction of the maculae, that reversed after-effects would be found.

Adaptation. Considerable discussion, some of it acrimonious, has appeared on the topic of vestibular adaptation. Does it exist and, if so, what is its nature? There is little doubt that under many circumstances habituation to motion does occur, although the reasons underlying it are obscure and there may be some situations under which some of the indicators of habituation do not show large changes. Experimental investigations of habituation to rotation show that nystagmus can be eliminated by a series of trials over a period of several days. Similarly, with extended practice over a period of time, other effects of vestibular stimulation are reduced considerably, particularly all those reflex effects which accompany vestibularly induced nausea. That similar effects also occur in the otolith organs, as well as in the semicircular canals, is demonstrated by the habituation to the motion of the sea known as " sea legs " and by the disappearance of seasickness in many passengers after a few days.

That these effects are not psychogenic is demonstrated by the fact that the removal of one lobe of the cerebellum (and of *no other portion* of the CNS) gives immunity to motion sickness in dogs (19). Many dogs become violently carsick when being taken for an unaccustomed ride, particularly upon mountain roads. After several such trips in close succession, habituation occurs and, even though greatly excited by the prospects of a trip, they no longer become sick.

Are these habituation effects true adaptation in the sensory sense? They do not appear to be similar to other cases of true adaptation, most importantly because the effect is not immediate, it does not become less the more intense the stimulus, and spacing over a period of several days is necessary. If the data are compared to those for other changes in behavior, it will be seen that vestibular habituation resembles learning rather closely. However, there are some differences. After a period of disuse, no matter how great the " practice " preceding it, the usual effects of vestibular stimulation return; " massed learning " does relatively little good; finally, learning on a symbolic level is not possible.

Motion Sickness. Motion sickness is a problem to a large number of people. It, therefore, also becomes a problem to the owners of transportation facilities and to the armed forces, particularly the Navy and of late, the Army whenever it is forced to land men prepared for immediate battle. Motion sickness may be characterized by any or all of a number of responses, depending upon the degree of severity — blanching, becoming chilly, sweating, queasy stomach, headache, feeling of enervation, loss of appetite and retching. It is important to know both what the effective stimulus for these responses is and, if possible, what can be done to alleviate the symptoms. A number of lines of evidence indicate that the most effective stimulus is the vertical motion (the pitch of a ship) rather than the rolling or yawing. Experimental studies have shown that both duration and acceleration are factors. Waves of relatively long periods produced sickness in more people than did waves of short periods, but the total energy in the wave was also important.[20] [21] Anyone who has sailed a small boat in the open ocean knows that the danger of seasickness is greatest when there is little wind and a large swell, for then one goes forward very little, and goes up and down a great deal. The stimulation of the utricle is maximal under these conditions and the expected results occur. Sickness does occur when the semicircular canals are stimulated, as has frequently been demonstrated experimentally. In daily life, it occurs upon driving a car along mountain roads. Here, again, a long period is involved. In "normal," uncivilized life, with no mechanical contrivances to impose severe conditions, the vestibular apparatus works well — it serves its functions and causes no illness, for the normal head movements are brief and of short period.

There are a number of steps which can be taken to reduce discomfort from vestibular stimulation, in addition to the commonsense ones of having had plenty of sleep, a healthful diet, and having avoided excessive alcohol. Gradual habituation over a period of time is sometimes possible, enabling one to avoid severe symptoms by short exposures. Placing the head in such a position that the accelerations do not act in the vertical plane of the head (that is, lying down) reduces the effectiveness of the stimuli. Fresh air and the avoidance of odors is beneficial, as is keeping warm. The use of drugs which are reasonably specific for motion-sickness, dramamine, for example, is very helpful, particularly if administered before the sickness occurs. It remains true, however, that there are great individual differences in susceptibility. Some individuals were "washed out" of flight training because they could not become habituated to the motion of airplanes. Air travel apparently causes the greatest incidence of motion sickness, followed by boats, and then by automobiles. In general, those individuals who have a history of sickness in cars, on swings, roller-coasters, and the like, are the ones who will probably also be sick relatively readily on other conveyances. Fortunately, if sufficiently interested and persevering in attempts to become habituated, most people can control these symptoms in any reasonable situation.

Suggested Readings

1. Lissman, H. W., "Proprioceptors," *Physiological Mechanisms in Animal Behaviour*. Symposia of the Society for Experimental Biology, No. IV, 1950, Chap. 3, pp. 34–59.
2. Lowenstein, O., "Labyrinth and Equilibrium," *Physiological Mechanisms in Animal Behaviour*. Symposia of the Society for Experimental Biology, No. IV, 1950, Chap. 4, pp. 60–82.
3. Ruch, T. C., "Somesthesis and the Chemical Senses," *Annual Reviews of Psychology*, 1953, 4, pp. 111–136.
4. Wendt, G. R., "Vestibular Functions," S. S. Stevens Ed., *Handbook of Experimental Psychology*. New York: Wiley, 1951, Chap. 31, pp. 1191–1223.

10 | The Chemical Senses

The so-called chemical senses are relatively neglected in modern life. So much of our work is concerned with listening and looking; so much of our recreation depends on radio, television, movies, concerts, and plays. Few people are concerned with odors or tastes in their work — perfumers, tea-tasters, wine-tasters, air-conditioning experts, manufacturers of some food products, and the like. Fewer people are concerned with odors or tastes for aesthetic enjoyment — a few gourmets make meals an aesthetic experience and there are "smell" gardens for the blind. But most of us eat to stop hunger pangs and plant our gardens for visual appeal. Yet the chemical senses are interesting and highly enjoyable if one but takes the trouble to attend to them and to become sophisticated in their subtleties.

One of the fascinating aspects of the chemical senses is the paucity of knowledge concerning them and the continued expectation of being on the brink of great discovery. Even the elementary psychology student can share in this, because he can readily ask questions which we cannot answer in the present state of our knowledge. For example, he can ask why these senses are called the "chemical senses." And we answer that the stimuli for these senses are considered to be chemicals. If the student then complains that this is not very definitive and that there are many chemicals which do not stimulate any of these senses, we can only agree. If pressed further we must admit that we do not know what the important aspect is which makes some chemical compounds effective as stimuli, but must try each compound, as it is discovered or synthesized, to find out whether it is effective. We have some hunches as to the important aspects of the stimulus, but so far none of them has been experimentally verified. Moreover, we are almost certain that stimuli do not have their effect primarily by means of chemical reaction with substances in the receptor cells, so that the important reaction between stimuli and receptors in the chemical senses is not chemical at all. The chemical senses are so named for historical reasons and it is not inconvenient as long as one is perfectly clear as to the basis for it. They have many problems in common — purity and control of the stimulus; stimulation by the same substance, in some instances, with the concurrent difficulty of separating the experiences psychologically; the kind of reaction occurring between the receptor cells and the stimulus; and many more. These problems make it convenient to consider these senses together even though they may not really be "chemical" senses.

There are often considered to be three of these "chemical" senses: (1) the simplest and most primitive is called the **common chemical sense**; (2) **gustation** (taste) is more complex, psychologically at least; and (3) **olfaction** (smell) is highly complicated. These senses are not merely different aspects of a single sense department, but are as separate and distinct as vision and audition. There are three lines of evidence which lead us to this conclusion. (1) Psychological: the qualities are quite different — compare the experiences of the taste of vinegar, the odor of roses, and the bite of pepper. (2) Physiological: by taking advantage of disease or injury, or by the use of narcotics, such as cocaine, it has been possible to show that any one of the three senses may be eliminated, leaving the others undisturbed. (3) Anatomical: the pathways in the central nervous system are separate and distinct for each of the three senses. They should, then, be regarded as separate sense departments, which are grouped together for discussion only because of certain common problems mentioned above. But we must also recognize that they are often confused in casual experience and that the complex perceptions of flavor in food often involve the stimulation of all three senses as well as certain of the skin senses — pressure, warmth and cold — and proprioception from the tongue and chewing muscles. As everyone is well aware, food is "tasteless" when one has a bad head cold. "Flavorless" would be a better word, for the taste receptors are still functioning, as are the common chemical receptors. You can taste the sourness of lemonade but are unable to appreciate the "lemonyness"; you can feel the bite of pepper or chili, but the aroma of the food is gone. It is the sense of smell which is impaired, as you can quickly prove to yourself by holding your nose tightly and comparing the bare taste of vinegar and red table wine or apple and onion. In the following sections we shall separate flavor into its components and discuss each sense department in detail.

The Question of the Common Chemical Sense. The term "common chemical sense" refers to a sensitivity, found in the mucous membranes in man, to certain kinds of substances, such as pepper on the tongue or ammonia in the nose or eyes. It is sometimes considered to be distinct from the sense of pain in the nose and mouth, but recent evidence favors the view that it is the ordinary pain system which is involved.[1] The receptors are probably free nerve endings and the impulses travel via cranial nerve V, the trigeminus, to the same nucleus of the thalamus that receives pain fibers from the head region.

We do not know what aspects of chemical compounds are responsible for their ability to stimulate these pain receptors. Some common stimuli are: ammonia, onion, and chlorine, which have characteristic odors, but also stimulate strongly the pain receptors in the eye and nose, causing them to water; and spices such as pepper and ginger, which stimulate the taste receptors and also the pain receptors in the mouth.

One study of thresholds for a number of substances showed that the chemical-sensitive receptors in the eye are more sensitive than those in the nose, although they are much less sensitive than the olfactory receptors. Chloracetophenone, an early war gas, was the most powerful stimulus studied; for the receptors in the eye the threshold was found to be .0083 parts per million of air, and for the receptors in the nose the threshold was .021 parts per million of air.[2] Thresholds vary with the stimulus used and some substances have lower thresholds for the pain receptors than for the olfactory receptors. One of the desired features of war gases is that they should be capable of great damage to the organism at concentrations below the olfactory, or even the pain threshold, so that their presence is not detected until the damage is done. Such a substance is Lewisite, which smells like geranium, but the odor threshold is well above the threshold for the pain receptors and for the formation of blisters. Much of the work on thresholds for chemical pain has been done by agencies interested in mine safety, as well as in gas warfare. In mine safety, it is important to find odorous warning agents which do not harm or even temporarily incapacitate the miners, in order that they may detect dangerous leaks and escape in time, and much research is directed toward their discovery.

GUSTATION

The sense of taste is of interest to all of us in that it contributes much to our enjoyment of food (although other senses also cooperate). As evidence of this, we find that in old age, as the taste receptors become less sensitive, the appetite fails. This is a contributing cause of the serious nutritional deficiencies which are often found in old people and which are important factors in their characteristic lack of energy and lack of interest in their environment. Taste thresholds appear to double about every 20 years. The changes in the taste receptors may also be important to food manufacturers. For example, one advertising campaign for a new beverage was directed at young people. It failed miserably and the company was wise enough to attempt to discover why instead of simply hiring a new advertising agency. The facts of the matter were that the beverage had a slightly bitter taste, which young people, on the average, do not like, but middle-aged people find pleasant. It was then a simple matter to direct the advertising to the middle-aged group.

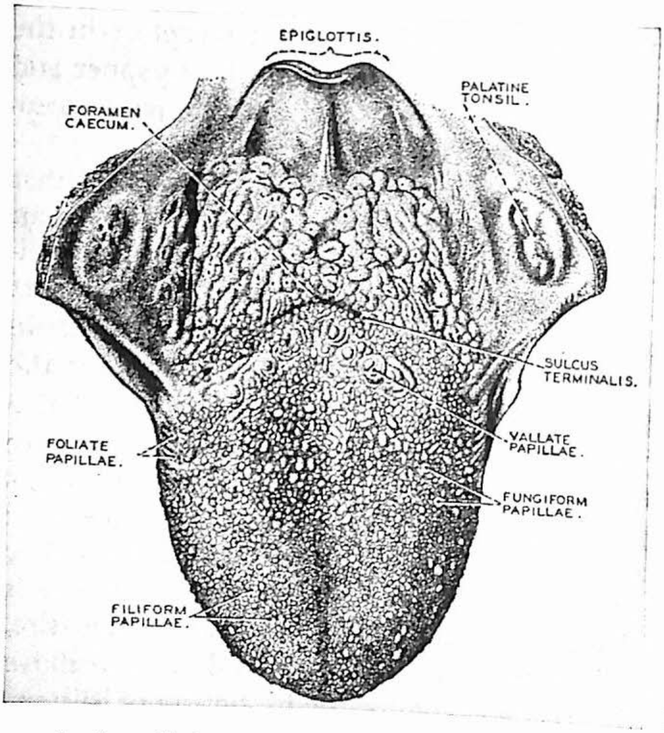


FIG. 10.1. Location of papillae on tongue. (From Neal and Rand.[26])

The Armed Forces are also greatly interested in gustation. To them it is vitally important in many ways that the food they purchase be acceptable to the men to whom it is served, and an important factor in food acceptance is the sense of taste. If the food is not acceptable, it means waste of a tremendous amount of money, waste of good raw materials, and, even more important, the men do not obtain the necessary nutrients for efficiency and alertness. It has been found in one of the branches of the Armed Services that about one-sixth of the calories provided per man per day, and significant amounts of protein and vitamins, find their way into garbage pails. So important is this problem that a large research program is maintained by the Armed Forces to find answers to both applied problems and basic problems in taste and flavor.

Receptors and Central Nervous Connections. The receptors for taste are specialized cells which are grouped together into **taste-buds** on the tongue, larynx, pharynx, and a few other spots. In children, they occur on the inner surfaces of the cheeks, but disappear in the adult. The taste-buds are most numerous in the **papillae** on the top of the tongue. Figure 10.1 shows the location of some of the papillae. The center of the tongue contains no taste buds and is, therefore, insensitive. Each taste-bud, which looks very much like a flower bud (Fig. 10.2), contains a number of taste cells, together with other cells which probably are immature taste-cells. The end of each taste cell sends forth a slender hair-like process which is probably the surface upon which the changes occur which lead to the discharge of the nervous impulse. There are no obvious morphological differences among taste cells to correspond to differences in taste qualities, so we assume that differences in cellular physiology underlie different taste qualities. Taste buds may be sensitive to only one or to several taste qualities.

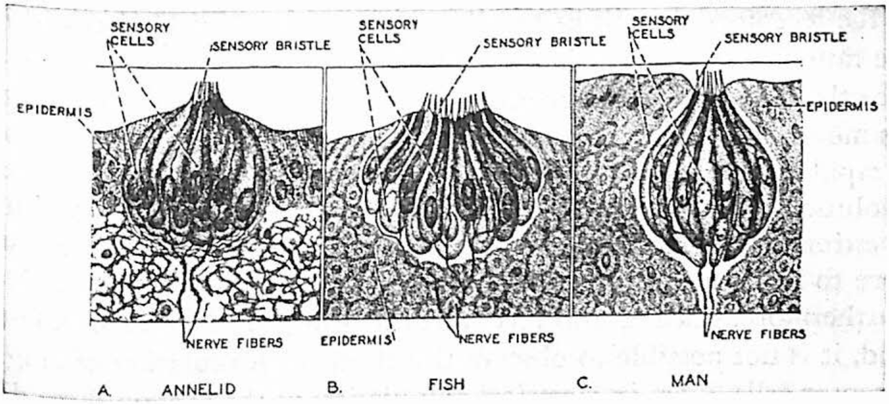


FIG. 10.2. Taste buds, showing taste cells, hairs, and pores. (From Neal and Rand.[26])

The afferent fibers which innervate the taste-buds run in two cranial nerves to the central nervous system. The posterior third of the tongue is served by fibers of the ninth cranial or glossopharyngeal nerve; the anterior two-thirds is served by fibers from the seventh cranial or facial nerve. Each nerve carries impulses which produce all the taste qualities.

These nerves run to the myelencephalon where the first synapses are made. Fibers then run to the thalamus, the exact location being uncertain. From the thalamus, projection fibers are sent to the cerebral cortex, terminating at the foot of the post-central gyrus, close to the general sensory mouth area and just behind the motor areas serving the chewing muscles. The cortical localizations for the functions dealing with the mouth are thus seen to be grouped rather closely together, which undoubtedly makes it easier to integrate all the diverse functions.

Experimental Difficulties. Before proceeding to a discussion of the stimulus and psychological attributes of this sense department, some consideration should be given to the experimental difficulties involved. One of the gravest of these arises from the inaccessibility of the receptors. As will be recalled, the receptors are buried within the taste buds, which makes it impossible to stimulate the receptor directly. The experimenter must either ask the subject to take into his mouth a quantity of solution containing a known concentration of sapid substance, or he must place a piece of cotton dipped in such a solution, or a drop of it, on the tongue. In any case, it is difficult to estimate how much of the substance actually penetrates the taste pore to reach a receptor and how diluted it has become by saliva. Furthermore, since several receptors are grouped together in a taste bud, it is not possible to observe the effects of stimulation of single receptor cells. Even in electrical stimulation of the tongue these difficulties are not avoided, for electric current probably stimulates, at least in part, by electrolysis of the saliva. Thus it may stimulate not only inadequately, by direct electrical stimulation of the end-organ, but also adequately by the formation of hydronium ions resulting from the electrolysis of the saliva. Hydronium ions are thought to be a stimulus for sour, and sour is the sensation which is usually reported when taste buds are electrically stimulated. In each of these cases it is the entire group of receptors within a taste-bud, or more likely in a number of taste-buds, which is stimulated. And here, also, the hydronium ion stimulus is diluted by an uncontrolled amount of saliva and the electrical conductivity may also be influenced by this factor. Where solutions of sapid substances are used, it is important to measure concentration very precisely and, also, to control the temperature of the solution, which ordinarily should be at physiological zero. In any type of stimulation, one must make sure that several hours have passed since the subject has taken food or drink and a thorough oral cleansing is in order before the start of experimental work, since adaptation changes thresholds.

Stimulus. We do not know how the stimuli for gustation act upon the receptor. We can give examples of effective stimuli, but we have not yet been able to discover a precise law which will enable us to predict the taste quality and threshold concentration of a substance, or even whether it will be sapid. We can, however, say that a substance will not have a taste of any sort unless it is appreciably soluble in water and, therefore, in saliva. In order to discuss intelligently the effective stimuli for taste, we must consider them in conjunction with specific taste qualities.

Qualities. It is usually agreed that there are four primary taste qualities and that all other tastes are a combination of these primary tastes. The four primary qualities are: sour, salt, sweet, and bitter. They are considered to be separate qualities for a number of reasons.

The typical stimulus for sour is an acid — hydrochloric acid, HCl, is an example. The H-ion is considered to be the effective stimulus, although other factors also enter to modify its effect, so that one cannot predict degree of sourness from knowledge of hydronium ion concentration alone. In addition, not all acids are sour.

The typical stimulus for salt is sodium chloride (NaCl), common table salt. Many of the salts of low and medium molecular weight taste salty. Apparently both the positive (Na^+) and negative (Cl^-) ions play a part in the production of the taste quality and even un-ionized molecules may also be important.

The typical stimulus for sweet is common table sugar or sucrose ($C_{12}H_{22}O_{11}$). But representatives of many classes of compounds are sweet. Saccharin is a substance which is often used as a sweetening agent by diabetics or by persons requiring a restricted caloric intake. Structurally it is quite different from sucrose, yet it is intensely sweet (about 700 times as sweet as ordinary sugar, if pure), even though it is but slightly soluble in water.

The typical stimulus for bitter is quinine. Caffeine is a common substance which is typically bitter, though less than half as bitter as quinine. But many classes of substances give rise to the bitter quality. We saw that most acids are sour — but picric acid is bitter. We saw that salts of low and medium molecular weight were salty, but salts of high molecular weight are often bitter. And there is a change

Table 10-1. Some Thresholds for Sapid Substances

[From Pfaffman, (14)]

Substance	Molar Concentration
Sucrose	2×10^{-2}
Sodium Chloride	3.5×10^{-2}
Hydrochloric Acid	2×10^{-3}
Crystallose (sodium salt of saccharin)	2×10^{-5}
Quinine Sulfate	4×10^{-7}

from sweet to bitter in many **homologous series** as the series is ascended.

Thresholds. Threshold measurements for the four taste qualities are rather variable, but it is interesting to compare their sensitivity. Table 10-1 gives the results of such comparison in terms of **molar concentration,** ranked in inverse order of sensitivity.

Let us now discuss the evidence for assuming there are four taste qualities. Introspective evidence is one line of evidence. When subjects are asked to describe the gustatory experiences obtained from a number of different solutions, with the sense of smell excluded, they are usually able to do so by reference to the four qualities cited above. You may roughly check this yourself by holding your nose as you taste various substances; black coffee tastes bitter, grapefruit tastes sour and bitter, lemonade tastes sweet and sour, and a salted cracker has an overtone of salt.

Another line of evidence comes from the differential distribution of the taste qualities on the surface of the tongue. The tip of the tongue is particularly sensitive to sweet, i.e., the thresholds are lower in this region than in other areas. The base of the tongue is especially sensitive to bitter. The sides show the lowest thresholds for sour. These taste qualities can be evoked by stimulation in other areas but the thresholds are higher. Salt is apparently about as easily sensed in one area as in another. These differences in distribution of sensitivity would argue for separate sensory systems. Further evidence of this is obtained by narcotizing the tongue with cocain; taste sensations disappear serially in the following order: bitter, sweet, salt, and sour.

There is also evidence that these taste systems are not completely independent, or at least that their interrelationships are complex. In one experiment, action-potentials were recorded from single nerve fibers of nerve IX in the cat, following stimulation with presumably sapid substances.[3] Only three kinds of fibers were found: (1) those responding only when acid stimuli were used, (2) those responding to both acid and sodium chloride, and (3) those responding to acid and quinine. In the cat, few responses to sugar were obtained, which may explain why cats do not like candy. Dogs like candy very much and they have fibers responding to sugar. The experimenter believes that these results are best explained by the assumption that the sensory quality depends not alone upon the excitation of a particular group of fibers, but upon a pattern of excitation within the total gustatory system.

Interrelationships among the taste systems can also be demonstrated by the change in sour threshold which occurs on the left side of the tongue when a subliminal solution of sucrose is placed on the right side. The formerly subliminal sour becomes immediately perceptible when the sucrose solution is applied on the other side.

Contrast, Compensation, and Fusion. It will be obvious from what has already been said that taste experiences are not usually simple. The phenomena of taste contrast, fusion, and compensation can be experienced in an ordinary meal — indeed, expert meal-planning takes these factors into account. Taste contrast may occur when substances having two or more tastes are placed in the mouth simultaneously or successively. For example, salt seems to increase slightly the sweetness of sugar. Eating salted crackers with ice cream is an unusual but very pleasant experience. Candy or sweet cake often makes tart fruit, such as grapefruit, taste more sour, which is equivalent to saying that partial adaptation to sweet enhances sensitivity to sour. On the other hand, after eating sour foods one requires less sugar in his coffee. Partial adaptation to sour might be expected to enhance sensitivity to sweet. This is taken advantage of in the Chinese dish, sweet-and-sour spareribs. Compensation may also occur among certain tastes. Salt and sour appear to depress each other, so that a salted grapefruit tastes less sour and less salt than one would expect. Taste fusion, in which individual tastes merge, is probably never complete. Although tastes do blend to some extent, it is ordinarily possible to analyze out the various components, particularly if odor is eliminated. For example, the bitter, sweet, and salt in cocoa form a nice blend, but it is possible, with practice, to taste the components. Also, one can obtain a reasonably good fusion of bitter and sweet in coffee, but the components can be recognized.

Adaptation. As we already know, adaptation is a very widespread phenomenon in sensation. For taste, we find that adaptation of single papillae and small areas of the tongue is complete. For example, one adapts to a 5-percent salt solution on a small area of the tongue in a very few seconds, and, after 30 seconds adaptation, recovers in about 30 seconds. Stronger solutions require more time for complete adaptation, and slightly longer periods for recovery. Complete adaptation for large areas does not occur in the normal course of events, not even during the course of consuming a large lollipop. Experimentally, however, it has been found possible to attain complete adaptation for the entire mouth for all four taste qualities. Table 10-2 gives the average time in seconds for complete adaptation to each of several concentrations (weak, medium, and strong) of the four stimulating substances. There are many difficulties in such experiments, but we may be confident from these results that, given constant stimulating conditions, taste adapts completely. Duration, as a psychological dimension, is thus correlated with the duration of the stimulus, but the intensity gradually decreases with continuing adaptation.

Table 10-2.

[From Dallenbach, et. al. (15)]

Taste Modality	Substance	Average Adaptation-time for 5 intensities of stimulus				
		1	2	3	4	5
Sweet	Sucrose	46.5 (0.066M)	90.5 (0.131M)	132.2 (0.246M)	193.1 (0.737M)	326.0 (1.260M)
Salt	Sodium Chloride	16.2 (0.84M)	73.3 (1.66M)	90.5 (2.44M)	115.6 (3.11M)	—
Sour	Tartaric Acid	78.6 (0.0022M)	129.6 (0.0066M)	194.8 (0.013M)	—	—
Bitter	Quinine Hydrochloride	74.7 (0.000013M)	115.3 (0.000041M)	115.6 (0.000083M)	—	—

Intensity. Gustation shows intensive as well as qualitative changes. In general, intensity of sensation increases as concentration increases, but some substances are much more effective stimuli than are others, so any comparison among substances must be done on an empirical basis. One study of perceived intensity for various concentrations of sodium chloride gave Weber fractions which varied from 0.24 to 1.25. Figure 10.3 shows the curve which results when $\frac{\Delta I}{I}$ (the Weber fraction) is plotted against I (the value of the standard stimulus) for this experiment. If the Weber fraction were constant, the graph would show a horizontal straight line. Although it deviates from this at the extremes, in the middle intensities the curve approximates a horizontal line. In general, then, the DL is proportional to the magnitude of the stimulus.

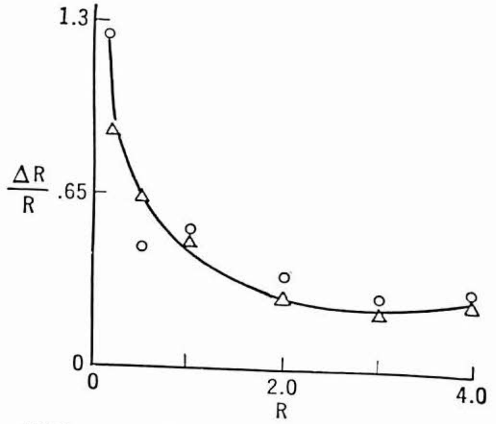


FIG. 10.3. Weber ratio for salt solution. (From Holway and Hurvich.[27])

Affect. It is of interest to note that the affective component of gustation is closely related to intensity. When weak, all the taste qualities are pleasant to many people, particularly adults with "cultivated tastes." Slight bitter is pleasant in coffee and beer, slight sour is pleasant in " dry " wine or lemonade, mild sweet and salt are almost universally pleasant. But all qualities, even sweet, become unpleasant if very strong. There are, in addition, age differences in likes and dislikes — children are renowned for their desire for sweets, whereas adults often come to prefer the bitter and sour qualities.

Effect of Temperature. Temperature has an important effect on the taste of various substances, and one which is not predictable from the simple chemical fact that substances are more soluble at higher temperatures. One study found taste sensitivity to increase when the temperature of the solution is raised from 10° to 20° C, as one would expect, but the sensitivity remained fairly constant from 20° to 30° C, and then decreased.[4] Another study found this effect only for salt and bitter, but found sensitivity to sweet and sour to increase continuously with temperature; [5] whereas still another found only sweet to rise and fall.[6] The fact that any of the taste systems operate contrary to prediction from our knowledge of chemical reactivity has important implications for theories of taste. In addition, these findings are of interest in everyday situations. For example, if soup is salted when it is very hot, it will be too salty by the time it is served, since sensitivity to salt increases as the temperature falls.

Deficiencies. Although there are individual differences in taste sensitivity, as there are in all other psychological functions, partial taste-blindness (**ageusia**) for an entire quality has not been reported. Whether this is due to some intimate connection among the taste systems so that they remain or are lost together, or whether such occurrence would not be noticed readily (because of the more important contribution of odor and other senses in the flavor of food) is difficult to determine. One taste deficiency for a synthetic substance, phenylthiocarbamide (PTC), has received much attention from geneticists because it seems to obey simple Mendelian laws of inheritance. The taste thresholds for PTC show a **bimodal distribution** — a large number of people are rather insensitive to it and are called nontasters.[7] About 30 percent of people are nontasters. To the rest, the substance is bitter. But this disability is specific to PTC and some very closely related substances; it does not affect the thresholds to unrelated bitter substances. The thresholds for quinine, for example, are uncorrelated with those of PTC.[8]

Theory. It is not profitable to discuss particular theories of taste stimulation because no theory has been proposed which is generally accepted, but we have already indicated certain experimental findings which set definite limits to theorizing. First, we have seen that chemical groupings do not permit prediction of taste quality except within rather wide limits. Second, we found that gustatory sensitivity, at least to salt and bitter, decreased with increasing temperature above 30° C. We are thus forced to conclude that a simple chemical theory of taste is untenable. This has the advantage of forcing a change in the direction of gustatory research which should result in information of a sort calculated to throw light on other phases of the problem. At present, we can say only that promising leads appear to be the measurement of the size of molecule which can pass through the cell membrane and measurement of effects resulting from the adsorption of sapid molecules on the cell membrane. Thus useful information may come from the new research in cellular physiology.

OLFACTION

Olfaction is a more complex sense than taste, and although much research has been reported throughout the years we are still far from a solution to even the simplest problems. As we have already indicated, odor contributes much to our enjoyment of food, for it is one of the more prominent aspects of flavor. But there are many other areas of everyday living in which smell is of great importance. A good sense of smell is an important safety device and we all use it to good advantage in detecting escaping gas, or a fire somewhere in the house. The sense of smell can be used as a safety device for the aircraft pilot also. A substance may be rubbed on the edges of the oxygen mask so that if the pilot smells it he knows his mask is leaking and can correct the difficulty before he blacks out. Unpleasant odors in the house cause enough concern so that a good deal of money is made from the sale of substances designed to eliminate them. Unfortunately, many of these substances have their effect by partially anesthetizing the olfactory receptors, so that one not only does not smell unpleasant odors but also does not smell flowers or good food and might also be unable to detect escaping gas. An important aspect of air-conditioning involves the elimination of "stale" odors, for it is the odor of "stale" air, not the oxygen content, which makes people feel as though they did not have sufficient air to breathe. It is obvious, then, that deodorizing or re-odorizing old, warm air is more economical than bringing in fresh, cold air. The same problem is an important one in submarines and in enclosed spaces in ships, and, indeed, the Navy has supported basic research in olfaction because of the importance of these aspects for the efficiency of personnel. Another phase of olfaction has been the basis for a tremendous industry — perfume. Millions of dollars are spent each year for perfumes, toilet waters, and perfumed soaps in order that women (and some men, too) may perfume themselves with the odors of flowers. Perfumes of various sorts are also added to many other products including cosmetics, cookies, candy, writing paper, and lingerie. It has been found that women's stockings sell better if slightly perfumed, and used cars that are given that new-car odor bring a higher price. The wine and liquor industries are likewise dependent upon the sense of smell and are doing basic research in order to find out more about the production of the desired flavor (largely odor). Of some importance was the odor-training given some commandos during World War II. This training was an important aspect of getting one's bearings at night. The trainee was required, for example, to go past the field of new-mown hay, to the left of the cow barn, and so on. A lighter note is added by the fact that one of the standard test odors is said to be called "submarine sailor."

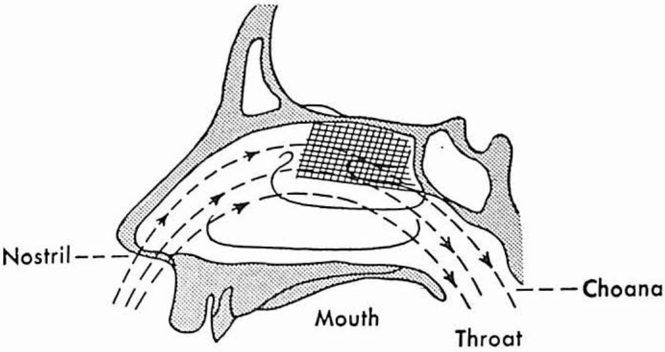


FIG. 10.4. Structure of the nose, showing location of olfactory epithelium (cross-hatching) and path of inspired air (dotted lines). Transverse view: the three curved lines represent the three turbinate bones. (Modified from Proetz.[28])

Receptors and Central Nervous Connections. The receptors for olfaction are bipolar cells found in the olfactory membrane among yellowish supporting cells. This **olfactory epithelium** is located high in the nose and covers an area of about 750 square millimeters on each side. Figure 10.4 shows the general structure of the nose, the location of the olfactory membrane, and the path of inspired air. It should be noted that this path brings the air, with its odorous molecules, to the lower reaches of the olfactory membrane, so that ordinary breathing results in the perception of odor. A vigorous sniff throws the stream higher, so that more of the olfactory membrane is bathed by odorous air and a more intense odor is perceived. The olfactory membrane, like the rest of the lining of the nose, is covered with mucus.

The olfactory receptor cells carry hairs on their outer ends. Some of these are long and may project through the mucus; others are short and are always covered with mucus (Fig. 10.5). The millions of olfactory nerve fibers stream upward through the bony floor of the skull in some 20 bundles, as cranial nerve I, the olfactory nerve, to end immediately in the olfactory bulb. The olfactory bulb, really a part of the brain, can be seen in Figure 4.3. There has been much dispute about the connections of the olfactory system within the brain. The sense is peculiar in that it does not have a major relay station in the thalamus. It is connected with a very old part of the brain, a part much older than the cortex of the visual or auditory or somesthetic systems, the anterior part of the pyriform area, which lies in the region anterior and ventral to the hypothalamus in Figure 4.2.

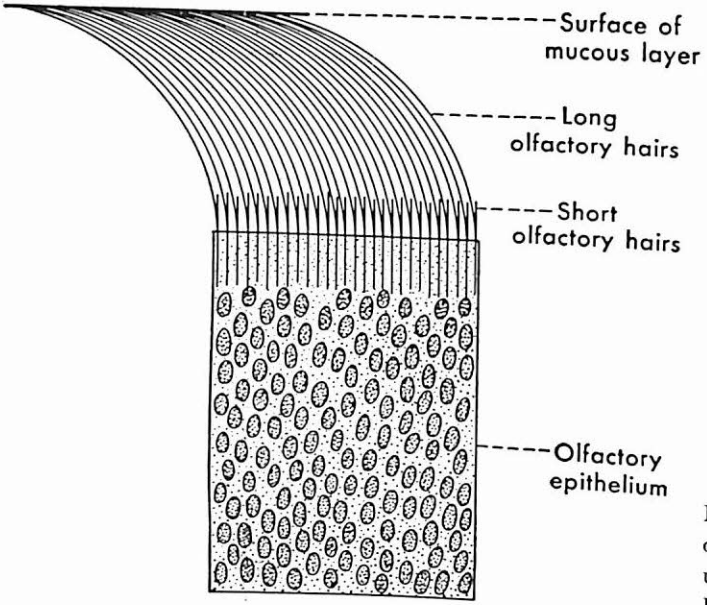


FIG. 10.5. Section of olfactory epithelium of frog. (From Hopkins.[29])

Experimental Difficulties. There are great difficulties involved in experimentation in olfaction and they may be discussed under three major headings. First, there is the problem of the purity of the stimulus. The sense of smell is so acute that a subject is often able to detect impurities which are undetectable by chemical tests. Most commercially available chemicals are not suitable for olfactory research. Second, there is the problem of the location of the receptors. They are so inaccessible that the best one can do about stimulating them is to introduce some odorous air at the nostril and wonder how much of it arrives at the olfactory membrane. The stimulus is always mixed with a variable amount of air, dependent partly upon the size of the nasal cavity, and the path taken by the airstream is determined in part by the irregularities of the nasal chamber and the vigor of the sniff. It is impossible to introduce a tube into the nose without causing distraction, sneezing, and possible damage. The stimulation of other sense departments in the nasal cavity, particularly cold and pain, by many odorous substances, makes the determination of the odor threshold difficult. Finally, the third problem is that of the control of the stimulus itself, which requires precise control of temperature, pressure, volume, and velocity, together with the maintenance of odorless air for the subject to breathe, and clean apparatus. This is an heroic task where the precise control of small quantities of gas are concerned. The fact that the difficulties are so numerous accounts for fact that we find little agreement among results.

Stimulus. As we have already indicated, we do not know what the stimulus is for olfaction. Some molecules of an odorous substance must come in contact with the olfactory cells — whether with the hairs or the cell body itself is unknown. But what process takes place there which results in the depolarization of the cell and the initiation of the nerve impulse, we do not know. It may well be that different kinds of substances stimulate in different ways. Every attempt to relate a specific kind of odor to a specific chemical structure has failed, so that at present we can only describe some general characteristics of odorous substances.

Few of the chemical elements are odorous — only the halogens (chlorine, bromine, iodine, and fluorine), yellow phosphorus, ozone (O_3), and arsenic. Of the inorganic compounds, a few are odorous, and they are mostly those which do not involve metallic elements. Some familiar examples are ammonia, and hydrogen sulfide — the latter is the active ingredient in the odor of rotten eggs. Most odorous substances are organic compounds, and there are tremendous numbers of them in all classes. Some examples are benzene, menthol, formaldehyde, ionone, and pyridine. But compounds with similar structure may smell very different and compounds with totally different structure may smell similar.

It has been said that, in order to be odorous, a substance must have: (1) an appreciable vapor pressure, (2) slight solubility in water, and (3) slight solubility in fat. But the necessary degree of each of these is slight, indeed, and, even if necessary, these conditions are not sufficient. The decisive factors in stimulating the receptors and the factors responsible for all the vast array of different odors still escape us.

Qualities. If you will reflect upon all the different smells you have ever encountered, you will realize how complicated the problem of olfactory qualities is. Each one of us has smelled thousands of different odors, even without entering an organic chemistry laboratory, where more thousands may be found. Certainly there is no immediately obvious classification of all these odors, such as we find with taste qualities or visual qualities. Many attempts have been made to bring some kind of order to olfactory qualities. Of these the most widely quoted have been three, all based upon the traditional introspective method which has worked so well in other sense departments. In 1895, Zwaardemaker, a Dutch physiologist, proposed a system of nine classes. In 1916, Henning, a German physiologist, proposed a system of six classes. And in 1927, Crocker and Henderson, Americans who were interested in using an odor classification for industrial purposes, introduced a descriptive system based

Table 10-3. Three Systems of Odor Classification

Author	Classes of Odors	Examples of Each Class
Zwaardemaker (16)	1 — etherial	fruit, acetic amyl ether
	2 — aromatic	camphor, citral
	3 — fragrant (balsamic)	vanillin
	4 — ambrosial	musk
	5 — alliaceous	onion, mercaptan
	6 — empyreumatic (burned)	phenol, pyridine
	7 — caprylic (hircine)	strong cheese, caprylic acid
	8 — repulsive	some nightshades
	9 — nauseating	feces, skatol
Henning (17)	1 — flowery	heliotrope, coumarin
	2 — fruity	fruits, citral
	3 — putrid	hydrogen sulfide, mercaptan
	4 — spicy	clove, anisaldehyde
	5 — resinous	turpentine, pinene
	6 — burned	tar, pyridine
Crocker and Henderson (18)	1 — fragrant	citral ($\underline{6}645$)*
		methyl salicylate ($\underline{8}453$)
	2 — acid	cineole ($5\underline{7}26$)
		20 percent acetic acid ($3\underline{8}03$)
	3 — burned	thujone ($66\underline{6}5$)
		guaiacol ($75\underline{8}4$)
	4 — caprylic	anisole ($257\underline{7}$)
		2,7 dimethyl octane ($351\underline{8}$)

* The order of the rating is the order for the classes as given.

upon four classes, each with nine possible steps, every odor being given a number from 0 to 8 in each dimension. These systems, with representative substances for each class, are presented in Table 10-3. They are presented here because they are so widely quoted and the problem is of great interest. Unfortunately, none of these has stood the scientific test, that of independent verification by other investigators. The only reasonable conclusion to be drawn from this is that the introspective method is not useful for this problem. In order to convince yourself of the difficulties of introspective classifications, try to order in some way all the odors you can find about the house.

Possibly relationships among odors will be discovered by one or more of three other techniques which are being used in research today: (1) studies of partial **anosmias,** directed towards discovering which substances are related by virtue of their all being either smelled or not smelled by the same individual;[9] (2) selective adaptation, in which adaptation is produced for one odor, and the effect produced upon other substances is measured, in order to see what relationships exist among a large number of substances;[10] and (3) statistical analysis of individual differences in sensitivity, in order to determine related groups of substances whose thresholds are always high or low together.[11] Data are not yet available for a sufficient number of substances to enable us to draw any conclusions at the present time. So we must simply state that there is no adequate classification of odors; that we do not know how many separate odors there are, nor how many classes; and, worse yet, we do not even know whether there are classes or whether odor qualities are spread out in a broad band, like the scale of pitches, without sharp groupings. It is usually assumed that there are a number of different kinds of receptors, to account for the variety of qualities, but there is little evidence for this.

Thresholds. Thresholds vary tremendously from substance to substance, as you might expect from the casual observation that a little perfume goes a long way, whereas it takes a sizeable sniff of orange juice to produce any odor. It is really not possible to compare thresholds for gustation and olfaction because we do not know how many molecules reach the receptor, but olfaction is ordinarily considered a much more delicate sense. The olfactory threshold for xylene musk was found in one study to be 5.9×10^{-12} (molar ratio). The sensitivity of olfaction is very impressive until one calculates how many molecules are probably involved in producing a threshold odor for a powerful substance like ethyl mercaptan — it is somewhere in the neighborhood of 1×10^{10} molecules (100 billion), although no great proportion of these reach the olfactory cells. This is not impressive when compared with 2 quanta as the threshold for the rods in vision. Table 10-4 presents a few representative thresholds for odorous substances. They provide some idea of the variation among substances and the relative order of magnitude of olfactory thresholds, but they are not strictly comparable, since the methods used to obtain them were different. Table 10-5 shows the wide variation in threshold which occurs due to differences in the degree of control of the stimulus and the purity of the substance.

Table 10-4. Some Representative Odor Thresholds

Substance	*Odor*	*Threshold (mg. per l)*	*Reference*
Carbon tetrachloride	sweet	4.533	19
Methyl salicylate	wintergreen	.100	19
Amyl acetate	banana oil	.039	19
N-butyric acid	perspiration	.009	19
Benzene	kerosiney	.0088	20
Safrol	sassafras	.005	21
Ethyl acetate	fruity	.0036	22
Pyridine	burned	.00074	2
Hydrogen sulfide	rotton eggs	.00018	2
N-butyl sulfide	foul, sulfurous	.00009	2
Coumarin	new-mown hay	.00002	2
Citral	lemony	.000003	23
Ethyl mercaptan	decayed cabbage	.00000066	2
Trinitro-tertiary-butyl xylene	musk	.000000075	2

Table 10-5. Thresholds for Methyl Salicylate

Threshold (Molar Ratio)	Author	Method
2.6×10^{-6}	Jones (24)	controlled volume and rate
1.9×10^{-5}	Gundlach and Kenway (25)	sniffing
11.5×10^{-5}	Allison and Katz (19)	sniffing

The differential threshold varies somewhat with the absolute intensity, as one would expect from other studies of Weber's Law, but a Weber fraction of .20 for the middle range is not far off. This is of the same general order of magnitude as the Weber fractions found in gustation. There are two other intensity effects which are of interest. As in taste, very intense odors are unpleasant, which may account for the fact that some people find a very strong odor of some flowers sickening. The other intensity effect, of importance for theory, is that the quality of some odors changes with intensity. β-ionone, for example, smells like violets when weak, but becomes a coarse cedary odor when strong.

Adaptation. Common experience provides many examples of the adaptation of olfaction. If you walk into a garden, you are delighted by the marvelous odor, but if you remain long it gradually fades. The odors of cooking, which greet you as you enter your home at dinner time, soon lose their charm. And, providentially, the odor that assails you as you enter a chemistry laboratory also loses its power. All this indicates that olfaction adapts rather quickly. The rapidity with which adaptation occurs depends upon the intensity of the adapting odor; very faint odors adapt in a whiff or two, but moderately strong ones may take 10 minutes or so. But adaptation is not an all-or-none affair; it is selective. Adaptation to one substance may affect the thresholds for other substances in several ways. It may raise the threshold enormously; the limiting case here would be the threshold for the adapting substance itself. It may, on the other hand, leave the threshold changed but little, or not at all. Or it may have an effect anywhere between these two extremes. The amount by which the threshold of one substance is raised by adaptation to another is taken as a direct measure of the degree of relationship between the two substances. This is the basis for the method, mentioned earlier, of investigation of olfactory qualities by selective adaptation. Vanillin and terpineol, for example, effect each other's thresholds greatly, but neither of them affects the threshold for butyric acid very much. Vanillin and terpineol are thought to be closely related in some important way, therefore, and to differ in some significant way from butyric acid. Much to everyone's surprise, it has also turned out that two substances which smell very similar and have a musk odor, do not lower each other's thresholds. It is possible that they excite different kinds of receptor cells, although they may have different effects upon the same cells.

Adaptation also results in a change in quality for some substances. Trimethylamine, for example, which smells fishy at the first sniff, comes ultimately to smell like ammonia. When this change in quality occurs, one assumes that certain kinds of receptors have been fatigued first, leaving the other elements in the complex less affected.

There are no after-images in olfaction.

Anosmia. There are some individuals who are completely anosmic — they can smell nothing. This condition sometimes results from a blow to the head or face, and can also result from severe and long-standing nasal irritation. It is of less interest, however, than partial anosmia. There are said to be a number of kinds of partial anosmia and one method for discovering primary odors is based upon an investigation of them. Smell blindness for some of the mercaptans, like taste blindness to PTC, seems to be relatively specific in that other odors can still be appreciated. Some individuals whose olfactory mechanisms are intact are unable to smell because of blockage of the nasal passages. They can, however, smell certain substances injected into the blood stream.[12] Temporary decreases in sensitivity occur from such diverse factors as smoking, nasal congestion, water in the nose from swimming, and exposure to a general anesthetic, such as ether. There is another kind of anosmia which may prove even more interesting because it is apparently linked to a hormone. The threshold for exaltolide seems to be related to the level of a female hormone (estrogen) in the body; 50–60 percent of adult males are completely anosmic to it and the sensitivity of sexually mature females fluctuates with the menstrual cycle, the threshold being lowest at ovulation.[13] Investigations of this sort may help us to discover the key to the stimulation of the olfactory receptors.

Theory. We are not poverty-stricken as far as olfactory theories go; on the contrary, we are overwhelmed with them. Unfortunately, there is very little evidence for any of them. It seems probable that there are several ways in which the receptors may be stimulated by different kinds of substances. It is certain that the answer will not be simple. At the present time the most promising leads derive from biochemistry and, particularly, the biochemistry of enzymes and drug action.

Suggested Readings

1. Moncrieff, R. W., *The Chemical Senses.* New York: Wiley, 1946.
2. Pfaffman, C., "Taste and Smell," S. S. Stevens Ed. *Handbook of Experimental Psychology.* New York: Wiley, 1951, Chap. 29, pp. 1143–1171.
3. Jones, F. N., and Jones, M. H., "Modern Theories of Olfaction: a Critical Review," *Journal of Psychology,* 1953, 36, 207–241.

11 Audition

That audition is an important avenue of information is obvious to everyone. With vision, it is one of the two important senses which give us knowledge of distant objects; unlike vision, it is useful around corners and in the dark. In addition, the complexity of possible patterning means that a great variety of information can be conveyed — for example, the almost infinite variations and combinations of sounds that occur in speech. Audition is one of the primary avenues of communication between people. One person gives another orders, or gives him information — as in a lecture, or by telephone, or in person. Much basic research on the nature of hearing has been done by agencies interested in the improvement of communication systems. The Bell Telephone Laboratories need such information as part of the basis for the improvement of telephonic communication, or The Armed Forces need adequate intercommunication systems in planes, tanks, and submarines, as well as field telephones. The problem is maximum intelligibility with minimum cost and one needs to know which variables may be sacrificed without significant loss, for faithful reproduction of sound (so-called " high fidelity ") costs money. Experimental work on sensory thresholds or thresholds of intelligibility is the business of the physiological psychologist and he is, thus, an important link in research programs such as those indicated above.

In addition to its importance as a means of communication, hearing is also of great significance in social relationships. People often talk because of feelings and not for the purpose of conveying information. They wish to establish friendly relationships, to express irritation, to win someone over. Much of our talk is " small talk " — gossip, table conversation — and it is important in human relationships; the person who does not have these skills to some degree has few friends. Convincing evidence of this may be found in the deaf and hard-of hearing, who often show radical changes in personality, becoming suspicious and irritable because they think people are talking about them. They also are often ill-at-ease in social groups and tend to withdraw more and more because they cannot cope with the subtleties of social intercourse based upon voice modulations. This leads us to problems of deafness and design of hearing aids.

Finally, the esthetic value of audition should be mentioned. Music, in all its many forms, is one of the most frequent sources of esthetic pleasure in many cultures. Not only is the simple melodic line pleasant, but great complexity of patterning is possible because of the analytical capacity of the auditory mechanism. For the sophisticated listener, the auditory patterns presented by a full symphony orchestra provide one of the highest developments of esthetic enjoyment. Part of our task is to inquire how such complexity of patterning is possible and what are the limitations of the auditory system.

THE STIMULUS

Sound Waves. The adequate stimulus for audition consists of sound waves in the surrounding medium, usually air. Since one of our problems is the relationship between the dimensions of the stimulus and sensation, our first task is the description of sound waves and the dimensions which the physicist uses for their specification. It is well to remember at this point that physical sound and perceived sound are not the same thing, even though, as we shall see, it is possible to predict one fairly well from the other. The legendary tree falling in the uninhabited forest no doubt produces sound waves — i.e., physical sound — but not perceptions. For the moment, our interest is in what the physicist has to say about sound waves.

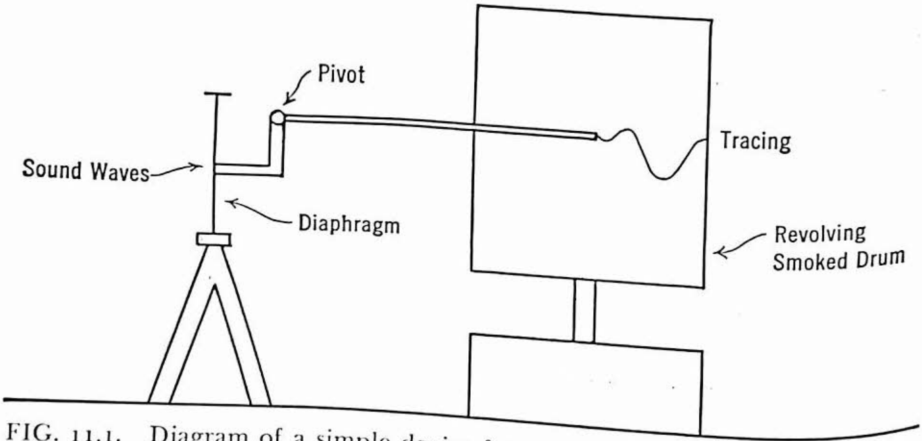


FIG. 11.1. Diagram of a simple device for recording sound waves. As sound waves push the diaphragm in and out, the lever moves up and down, tracing the pressure changes on the drum.

Sound waves are advancing regions of compression and rarefaction in some medium, such as air or water. These regions of compression and rarefaction are possible because the particles in the medium are separated by some amount of space — a good deal less in water than in air. As the waves advance, the particles within the medium travel back and forth along the same path as the advancing waves. No net progress is made by the particles; they simply move back and forth around the same point. As the particles move back and forth, they produce the regions of compression followed by regions of rarefaction which we have called sound waves. Figure 11.1 pictures an apparatus for recording sound waves in visible form. As the waves strike the diaphragm, they cause it to move back and forth. The movement of the diaphragm is transmitted to the lever, which in turn moves upward and downward, tracing the wave form on the revolving drum. The movement of the drum permits us to draw a line representing the movements of the air particles (upward and downward correspond to compression and rarefaction) through time. If the recording system is without mass or other source of distortion, a condition which we shall assume for the sake of explanation, the resulting curves will be, in the case of the simplest possible sound, what are called **sine waves.** A sine wave results if we plot along the horizontal axis the angles from 0 to 360°, as would be found in a complete circle, and the sines of the angles on the vertical axis. Hence, a complete wave, or **cycle,** contains 360° and the sine wave starts at 0, since the sine of 0° is 0, reaches a maximum at 90°, crosses 0 at 180°, reaches a minimum (or maximum negative value) at 270°, and returns to 0 at 360°. Of course, the wave goes on and on instead of stopping with one cycle. If we consider a given wave at a given moment, it will be at some point in a cycle. This point is called the **phase** of the wave at the given moment. It is important because two sounds otherwise identical may differ in phase, as we shall reiterate below.

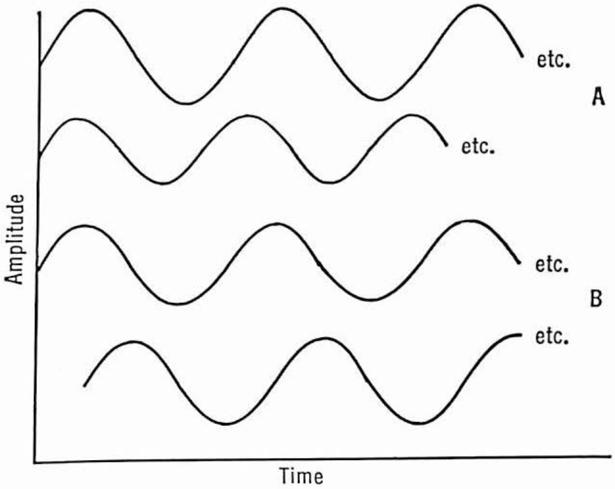


FIG. 11.2. The waves of pair "A" differ in frequency; those of pair "B" differ by 90° in phase.

A simple sine wave can differ from other waves in three ways. First of all, it may have greater or less **frequency.** The number of complete cycles in each second (*cps*) is the frequency. In Figure 11.2, the upper pair of sine waves differ in frequency. The second way in which sine waves can differ is in amplitude, or extent of the maximum displacement of the wave from the 0 line. This dimension of the wave is not as simply designated as frequency and so a separate section is devoted to it below. The third way in which two simple sine waves can differ is in phase. Two waves of the same frequency and amplitude may be at different points in their cycles at a given instant, either because they started at different times, or have traveled different paths. The difference in phase is designated by the degree difference in their cycles. In the lower portion of Figure 11.3 there are two waves differing only in phase, in this case, 90°.

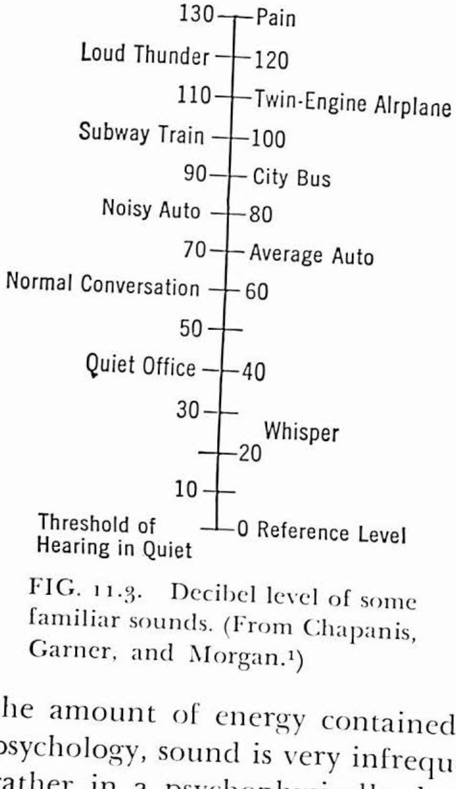


FIG. 11.3. Decibel level of some familiar sounds. (From Chapanis, Garner, and Morgan.[1])

The Decibel. The amplitude or magnitude of a sound wave may be designated in physical terms as intensity, or power. This refers to the amount of energy contained in the wave. In engineering and psychology, sound is very infrequently designated in such terms, but rather in a psychophysically derived term, the **decibel** (*db*). The decibel was devised in the Bell Telephone Laboratories as a psychologically meaningful way of describing the intensity of sounds. That the decibel is not actually a precise designation of the perceived loudness resulting from a sound of given intensity has not caused its abandonment, largely because it permits more compact plotting of sound intensities by reducing manyfold the length of the intensity scale.

Originally, the decibel scale started with its 0 point at the threshold energy for a given frequency. Thus, strictly speaking, each frequency and each individual required different intensity scales because the threshold varies both with frequency and from person to person. This is obviously very impractical, and nowadays the 0 is set at an arbitrary physical intensity level, ordinarily .0002 dyne per square centimeter (that is, 10^{-16} watt per square centimeter), regardless of frequency. Any sound intensity can be expressed as a ratio of this base intensity — it may be 10 times it, or 1,000,000 times it, or any other number depending upon the actual fact. The **bel** (10 decibels) is very simply derived from this ratio — it is the common logarithm of the ratio between the base or reference intensity and the sound whose intensity we wish to designate. Thus, the sound which has an intensity 10 times the base is at the level of 10 db, i.e., 1 bel, since the logarithm of 10 (the ratio between the sound and the base) is 1, and 10 times 1 is 10. A sound 1,000,000 times the base has a level of 60 db, the logarithm of 1,000,000 being 6. In order to make these levels meaningful, Figure 11.3 shows some common sounds at their proper points along a decibel scale.

Complex Waves. So far we have dealt only with simple sine waves which are sometimes called **pure tones.** These are rarely encountered. It is conventional, however, to describe a periodic wave as though it were made up of sine waves of differing frequency, intensity, and phase. When such an analysis is performed mathematically, it is called a Fourier analysis. That Fourier analysis has more than mathematical interest is demonstrated by the fact that it can be performed by mechanical and electronic analyzers as well as, more roughly, by the ear. Figure 11.4 shows a complex wave and the components obtained by Fourier analysis. In cases where the sound is not periodic, we speak of **noise.**

Sources of Sound Waves. Sound waves originate when some body vibrates, thus inducing compression waves in the surrounding medium. Examples are violin strings, drum membranes, the vocal cords, and tuning forks. When these are set into vibration in air, they impart their motion to the air particles around them, thus initiating sound waves. The frequency, intensity, phase, and complexity of the resulting sound waves will depend upon these same dimensions of vibration in the initiating object. A tuning fork vibrating at 100 cps gives rise to a sound wave of 100 cps, for example.

Harmonics. The tuning fork mentioned in the preceding paragraph vibrates in a simple manner, giving rise to sound waves which are very nearly pure sine waves. The violin string behaves quite differently. It vibrates not only as a whole, but also the various whole number fractions of the string vibrate separately. In the string each half, each third, each fourth, each fifth, and so on to each *nth* part vibrates separately. Since the frequency of vibration of a string of given mass and tension depends inversely on its length, it will be seen that the halves are vibrating twice as fast as the whole string, the thirds are vibrating 3 times as fast, and the *nth* portions of the string are vibrating *n* times as fast. In common terminology, the frequency of the whole string vibration is called the **fundamental** and the vibrations of the parts are called **overtones.** The first overtone results from the vibration of the halves and is thus 2 times the fundamental in frequency, while the second overtone is 3 times the fundamental. In general terms, the *n*th overtone has a frequency $(n+1)$ times the fundamental, e.g., the tenth overtone is 11 times the fundamental frequency, the twelfth overtone is 13 times the fundamental.

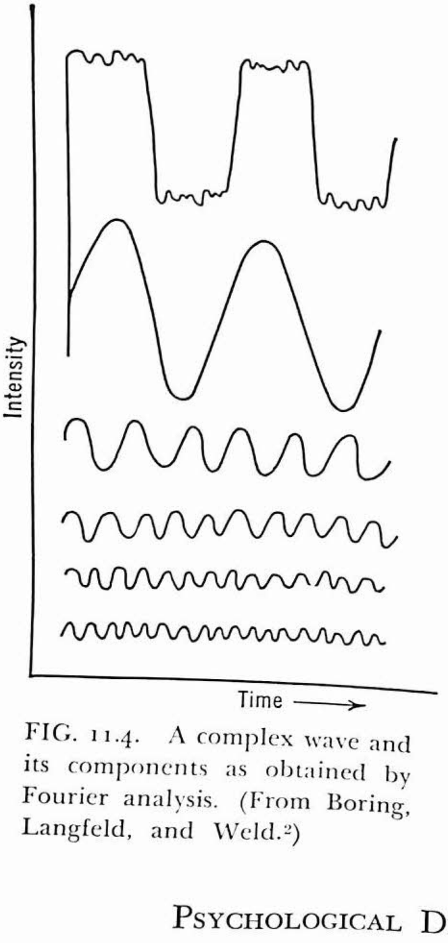


FIG. 11.4. A complex wave and its components as obtained by Fourier analysis. (From Boring, Langfeld, and Weld.[2])

Any body which has a single fundamental frequency of vibration has overtones which stand in the above relationship. If the fundamental is the same for two bodies, the overtones will therefore have the same frequencies. The overtones need not be of the same intensities, however, a fact which permits us to distinguish between two different instruments even though they are sounding a note of the same fundamental frequency. A piano and a violin playing the same note sound quite different because of the difference in intensity patterns of the overtones. Figure 11.5 illustrates the complex wave forms of some different musical instruments.

Psychological Dimensions of Sound

The dimensions of the sound wave are often erroneously used interchangeably with the dimensions of auditory sensation. We have avoided, for example, referring to the frequency of a sound as its **pitch,** or to the intensity of a sound as its **loudness.** These, with **timbre,** represent the most common dimensions use to describe auditory sensation.

Pitch is usually described as higher or lower and is most closely related to frequency of the stimulus. In general, sounds of greater frequency give rise to sensations of higher pitch. The relationship is qualified by the fact that an increase in intensity of a pure sound below about 4000 cps leads to a slight lowering of pitch, even though frequency is constant.

Loudness is closely related to intensity of sound, if frequency is held constant. If frequency is not held constant, the fact that the ear is more sensitive in the middle ranges of frequency will lead to changes in loudness as frequency is varied up and down between the extremes of hearing. Complete explanation of the point will have to await the discussion in the section on thresholds later in this chapter.

Timbre, or quality, is most closely related to the complexity of the sound wave. The sound of a violin, for example, contains rather strongly the overtones of lower number, while the sound of a trumpet contains relatively strongly the overtones of a higher number (Cf. Figure 11.5). The difference in timber is clearly perceived.

RECEPTORS AND CENTRAL NERVOUS SYSTEM CONNECTIONS

The physiological mechanisms underlying audition are extremely complex and we can consider here only a simplified version of them.

The Ear. The actual receptors for audition — the cells which serve to translate mechanical vibrations to nerve impulses — are the hair cells of the **Organ of Corti.** All other parts of the ear are accessory structures, strictly speaking, although they have extremely important roles to play in the process of hearing. In the discussion below, Figure 11.6 will serve to illustrate the various structures described.

If we follow the path of a sound wave as it enters the ear, we encounter first the **outer ear,** which consists of the **pinna** and the **external auditory meatus.** In man, the pinna has but little effect upon hearing. The meatus serves to conduct sound waves to the deeper structures and has an important effect upon the sensitivity of the ear because the air column in it is resonant, i.e., has a natural vibration frequency, at about 4000 cps.[1] This helps account for the point of lowest auditory threshold. Separating the outer and **middle ear** is the ear drum, or **tympanum.** Connected to the tympanum, and extending across the air-filled middle ear, are the three bones, or **auditory ossicles,** which serve to transmit the movements of the tympanum to the **inner ear.** The ossicles are named, beginning with the one attached to the tympanum, the **malleus** (hammer), **incus** (anvil), and **stapes** (stirrup). These ossicles form a lever chain which, with the tympanum, serve to reduce the amplitude of vibration while

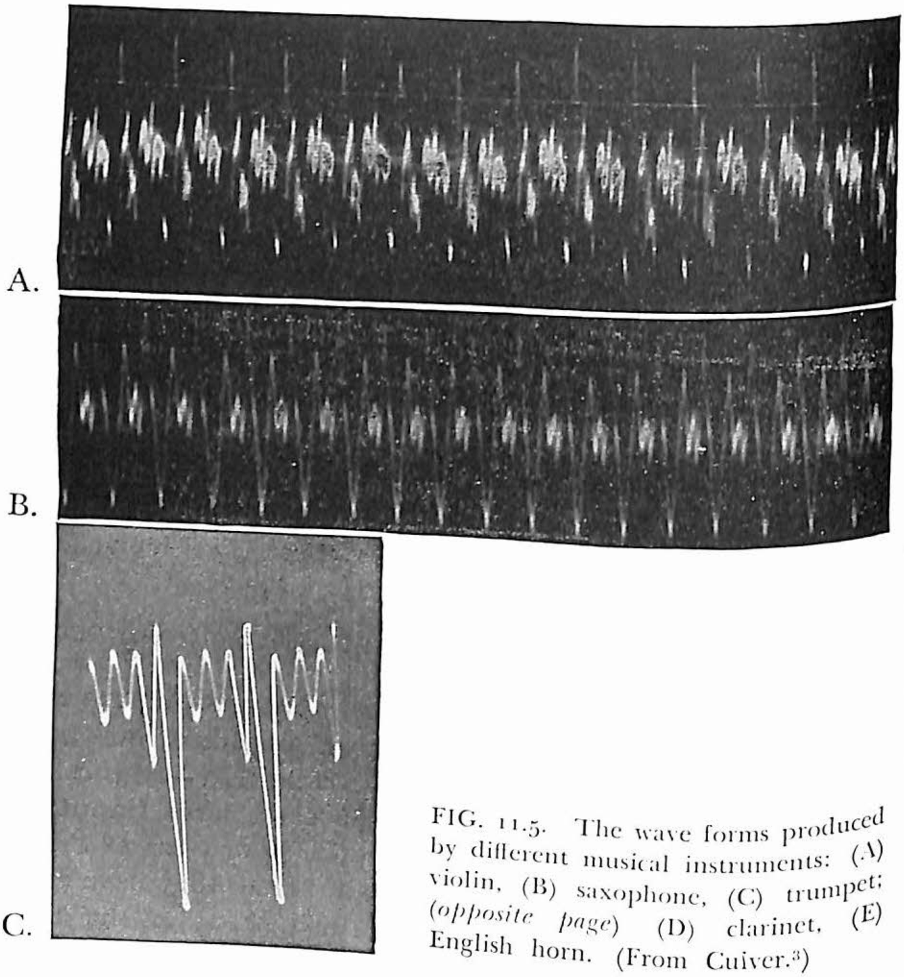
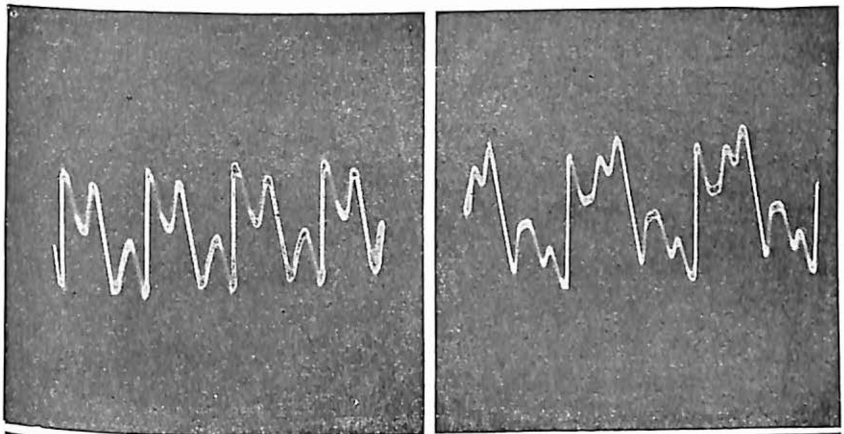


FIG. 11.5. The wave forms produced by different musical instruments: (A) violin, (B) saxophone, (C) trumpet; (*opposite page*) (D) clarinet, (E) English horn. (From Culver.[3])

increasing the force, the mechanical advantage being about 15 or 20 to 1. Connecting the middle ear to the throat is the **Eustachian tube,** which permits maintenance of pressure balance between the outside air and the air in the middle ear. Otherwise, changing external pressure could put severe strain upon the ear drum.

The stapes makes contact with the inner ear at the **oval window.** The inner ear, as has been noted in a previous chapter, has proprioceptive as well as auditory functions. The auditory portion of the inner ear is called the **cochlea,** which, with the rest of the inner ear, lies in the hardest bone in the body, the petrous. Essentially, the cochlea consists of a liquid-filled tube coiled 2½ times, down the center of which are two dividing membranes, the thin **Reissner's membrane,** and the relatively thick **basilar membrane,** which extend the whole length of the cochlea except at the very apex. These structures are visible in the cross section in Figure 11.7. The basilar membrane tapers somewhat from base (narrowest) to apex (widest),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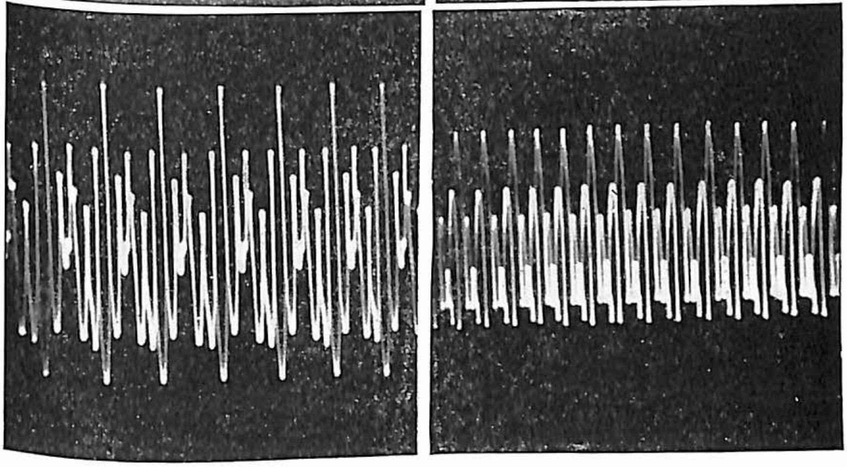

E.

a fact which is sometimes considered to be important for auditory theory. On the inner end of the basilar membrane may be seen the organ of Corti, composed of cartilageous supporting arches which hold the hair cells. Adhering to the hairs is the **tectorial membrane.** Stimulation of the hair cells (the actual receptors) occurs when the vibrations of the ossicles are transmitted through the oval window to the fluid, the **endolymph,** in the cochlear passages, thus causing a movement of the basilar membrane and the hair cells in relationship to the tectorial membrane. With each vibration of the basilar membrane, an alternating stretch and compression of the hair cells occurs, which serves to alter their membrane potentials. This alteration of hair cell potentials leads to the periodic stimulation of the fibers of the auditory nerve with which they synapse. The question of whether only a limited extent of the basilar membrane is affected by a sound of a given frequency or the effect of a given sound is widespread must await the discussion of auditory theory in the next chapter. The vibration of the endolymph (which is virtually incompressible) is made possible by the **round window,** which bulges in and out as the stapes moves out and in.

Nervous Connections. The primary neurons of the auditory nerve have their cell bodies in a ganglion within the cochlea itself. At their outer ends they make contact with the hair cells of the organ of Corti and at their inner ends synapse with the secondary neurons in the **cochlear nuclei** of the medulla. Figure 11.8 diagrams these relationships. It is of importance in auditory theory that each nerve fiber connects to many hair cells and that there is considerable branching of the nerve fibers resulting in cross-connections among them. Beyond the cochlear nuclei, there are four main destinations for fibers of the auditory tracts. First, there are the reflex and relay nuclei of

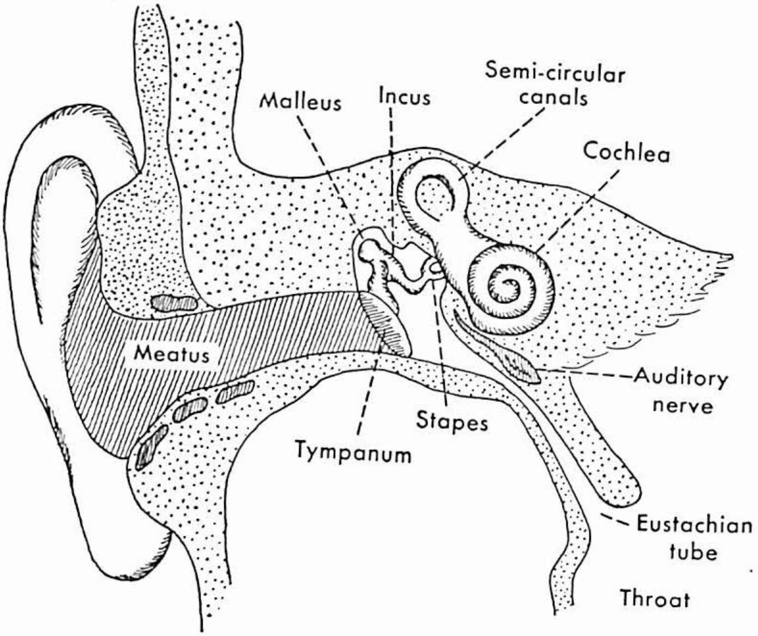


FIG. 11.6. Diagram of the human ear lying in place in the skull. (From Schaefer.[4])

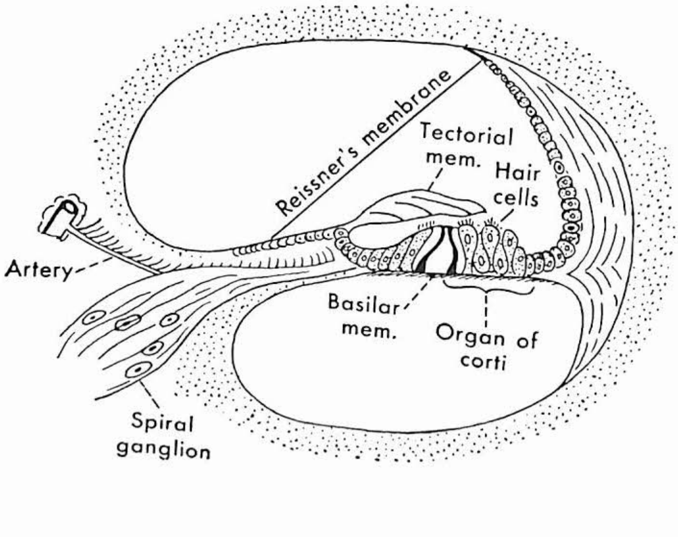


FIG. 11.7. Cross section of the cochlear canal. (From Davis.[6])

the lower brain stem. Secondly, there are collateral connections to the cerebellum, whose precise function is unknown. Thirdly, there are strong connections to the inferior colliculi and, by way of these, to the superior colliculi. Possibly these are important for orientation to sound. Fourthly, there are connections to the medial geniculate nucleus of the thalamus, which serves, in usual thalamic fashion, as a relay nucleus to the cerebral cortex. The auditory projection areas of the cortex lie along the upper edge of the temporal lobes. As is the case with somesthesis, the projection areas are at least two in number, although any difference in function between them has not been worked out.

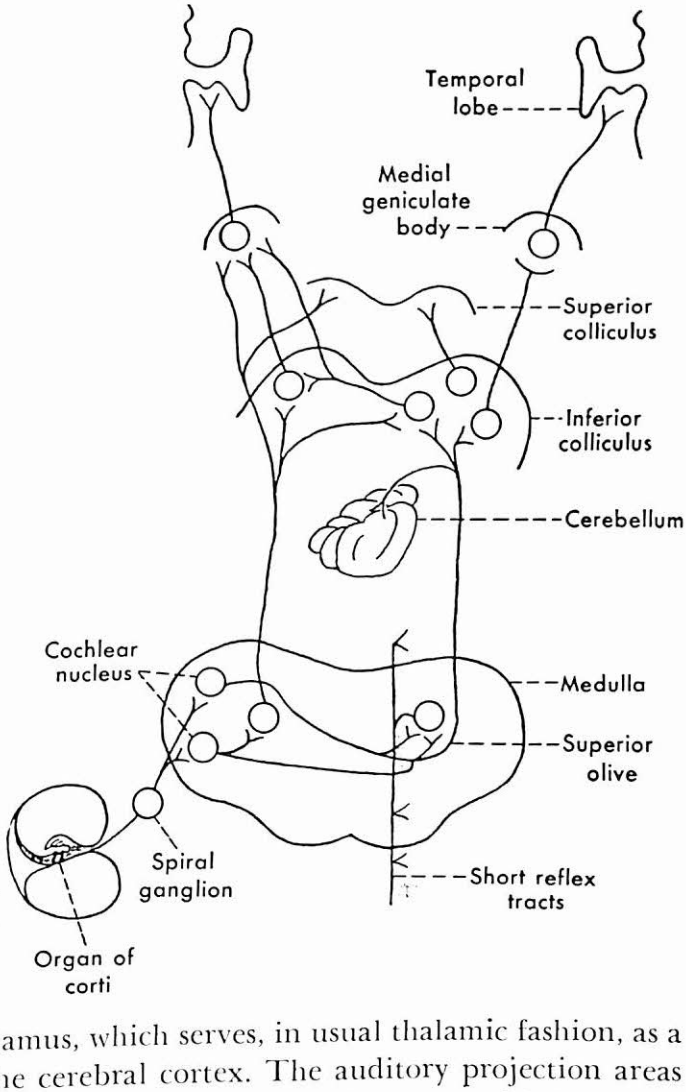


FIG. 11.8. Diagram showing the complexity of the connections of one ear. (From Davis.[7])

Of considerable importance are two further facts. First, the diagram in the figure represents the connections of one ear. As may be inferred, therefore, both ears have connections with both sides of the brain. Removal of one temporal lobe does not cause deafness in one ear, as would be the case if only unilateral connections were the rule. There is great opportunity for interaction between the connections from the two ears — a point of importance in the localization of sound. Secondly, the cortical projection areas show some differentiation according to the frequency of sound stimulating the ear. As may be seen in Figure 11.9, the two projection areas are alike in that different frequencies of sound at the ear lead to arousal of different regions but differ in the details of location.[2] This spatial differentiation according to frequency has been suggested as an important factor in pitch discrimination. The functions of the CNS connections will become clearer as our discussion of audition continues.

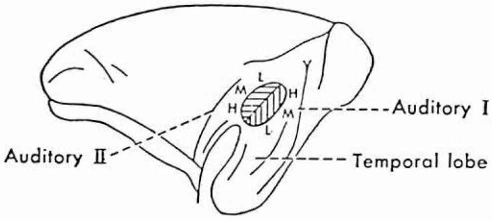


FIG. 11.9. The cortical auditory areas in the left cerebral cortex of the monkey. The letters H, M, and L refer to the cortical localization for high, medium, and low frequencies. (Redrawn from material in Woolsey.[8])

AUDITORY THRESHOLDS

Pitch: Absolute Threshold. There are frequency limits in man and other hearing animals above and below which a sound wave will not give rise to sensations of sound. The exact limits depend upon a number of factors, the most important of which are differences from person to person and the intensity of the sound. We shall confine our discussion to normal, young human ears for the moment and leave individual differences for future discussion. As for intensity, we shall assume that the frequency limits we are discussing refer to as intense a sound as can be produced without damage to the organism. Under these conditions, in man, the frequency limits are approximately 15 and 20,000 cps. The lower frequency may be called the RL for pitch, the upper frequency the TL for pitch. Especially at the lower end of the frequency scale it is technically extremely difficult to produce and lead to the ear a pure tone of sufficiently high intensity to be heard. Yet one experimenter has found the RL to be as low as 1 cps when he used a sound about 140 db louder than necessary to stimulate at 4000 cps.[3] At frequencies below 15 cps, however, what is heard is a series of chugging noises, no doubt arising from distortion in the ear itself. At about 15 cps and above, however, a continuous tone is heard and we, therefore, take this frequency as the RL for pitch. At the TL, threshold judgments are much easier. At some point, as frequency is increased, nothing is heard.

Pitch: Difference Threshold. The DL for pitch depends both upon the frequency of the standard tone and its intensity. In general, we may say that the Weber fraction is lowest for sounds of about 1000 to 2000 cps, and that up to an optimum intensity the louder the sound the lower the Weber fraction. In other words, a smaller proportion must be added to a frequency of 1000 to 2000 cps for the change to be noticed as a rise in pitch than is the case for frequencies higher or lower on the scale. Also, up to the optimum point, the ear is more sensitive to frequency changes in louder rather than in softer sounds. If your pitch is insecure, best sing softly! The Weber fraction for pitch at various frequencies is plotted in Figure 11.10. These data refer to a moderate intensity level. If we consider the DL in cycles per second rather than Weber fraction, we find that it is progressively smaller as we go lower in frequency. Figure 11.11 illustrates this point and, in addition, shows the dependence of DL upon intensity. In other words, at the lower frequencies the size of the DL is smaller, but it represents a relatively greater proportion of the standard.

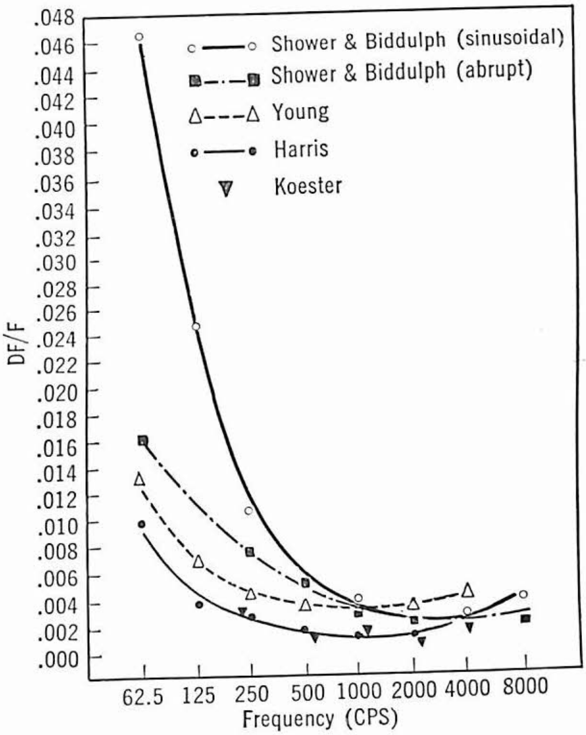


FIG. 11.10. The Weber fraction ($\Delta R/R$) for frequency discrimination of sounds of moderate intensity at different frequencies. (Redrawn from Harris.[10])

It is interesting to note that our musical scale assumes the constancy of the Weber fraction. The various intervals are frequency ratios, e.g., the octave is a 2 to 1 ratio, that is, a tone one octave higher than another is twice its frequency. Since the data indicate that the Weber fraction is not constant, it is apparent that the musical intervals are not exactly alike from octave to octave. Sometimes pianos are actually tuned progressively sharper at the high end of the keyboard, a device which makes allowance for the facts of Figure 11.10.

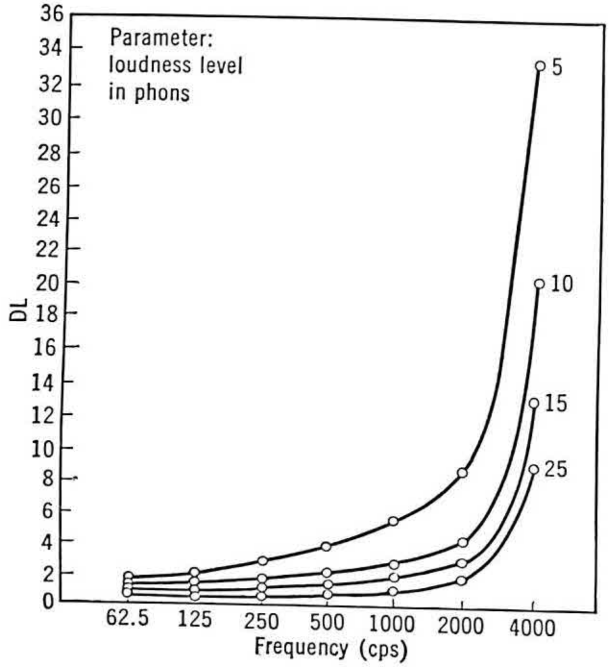


FIG. 11.11. Difference thresholds (DL's) for frequency given in cycles per second. Each curve represents the results obtained when the standard stimulus is at a given subjective loudness level. The term " phon " is a loudness designation: the higher the number, the greater the loudness. (From Harris.[11])

To make the DL's more concrete, it may be noted that at about 60 cps the DL for a moderately loud tone is in the neighborhood of 1/5 of a semitone (a semitone would be the difference between an adjacent white and black key on the piano) — at about 400 cps it is approximately 1/10 of a semitone. Modern music written on a scale of quarter-tones is well within the ability of the listener to discriminate.

Loudness: Absolute Threshold. The absolute threshold for sound depends upon the frequency. Beyond the frequency limits for hearing, there is no loudness. Over the frequency range which will evoke hearing, the threshold varies quite considerably, as reference to Figure 11.12 will show. The threshold is lowest between 2000 and 4000 cps and rises to comparatively very high levels as frequency is either raised or lowered. There are considerable individual differences in level of sensitivity, even among presumably normal, healthy young men. With practiced subjects these threshold measurements are extremely reliable and can be duplicated from day to day within 1 db.[6]

The astonishing sensitivity of the ear needs further comment. If we consider the distance through which the ear drum is vibrating at threshold (for a sound of about 3000 cps), we find that it is less than .000000001 centimeter, or less than the diameter of a hydrogen atom, and within the range of X-ray wavelengths. The basilar membrane vibrates over a much smaller distance than this — almost within the range of radii of the nuclei of atoms, or the wavelengths of the longer cosmic rays.[7] Is it surprising, then, that real silence never occurs for the normal subject? Even if all external sounds are removed from the surrounding air, a virtually impossible task, the sounds internal to the person himself — blood rushing through the vessels of the ears, sounds from respiration, even the creaking of joints — provide sufficient sound energy to exceed the threshold.

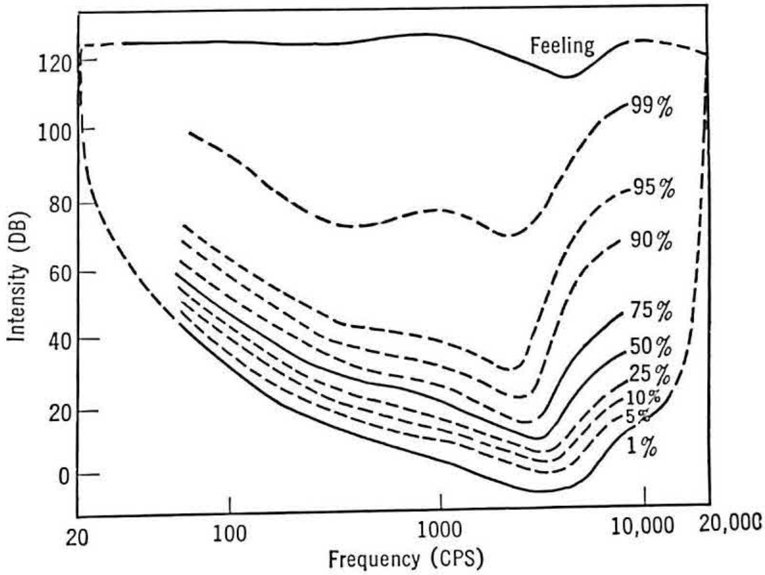


FIG. 11.12. Absolute auditory thresholds at different frequencies. Each curve shows the percent of the sample group which could hear a sound of the given intensity and frequency. (From Fletcher.[12])

Loudness: Difference Thresholds. Just as the absolute threshold for loudness depends upon frequency, so do the difference thresholds. Since the DL's vary with frequency and also (since the Weber fraction is not the same at all intensities) with intensity of the standard, it is customary to summarize these facts by plotting **equal loudness contours.** Reference to Figure 11.13 will make these clear. The lowest line represents the RL, and drawn above it are a series of lines representing sounds of the same loudness to the average observer. It is interesting to note how much farther apart the equal loudness contours lie in the middle frequency range as compared to the extremes of the audible scale. As a result, by the time rather high intensities are reached, the contours flatten and the same intensity gives rise to much more nearly the same loudness for all frequencies.

Considerable practical use of the plot of Figure 11.13 is made by sound engineers. As one applied example, we may consider a home music system. In the home, music is ordinarily played at a very much lower intensity level than the original. A symphony orchestra may be putting out 80 or more db across the frequency range. If the same music is reproduced in the home — from records or radio — at perhaps 50 or 60 db, the frequency extremes will sound very much less loud than the middle range. This fact can be predicted by drawing a line across the plot of equal loudness contours at the 50-db level and noting the contours crossed at the different frequencies. At this level, one can hear only the frequencies between 150 and 10,000 cps. The music lover's remedy may be one of two alternatives. Either the music must be played at its original loudness level or the bass and treble must be boosted to fit the contours.

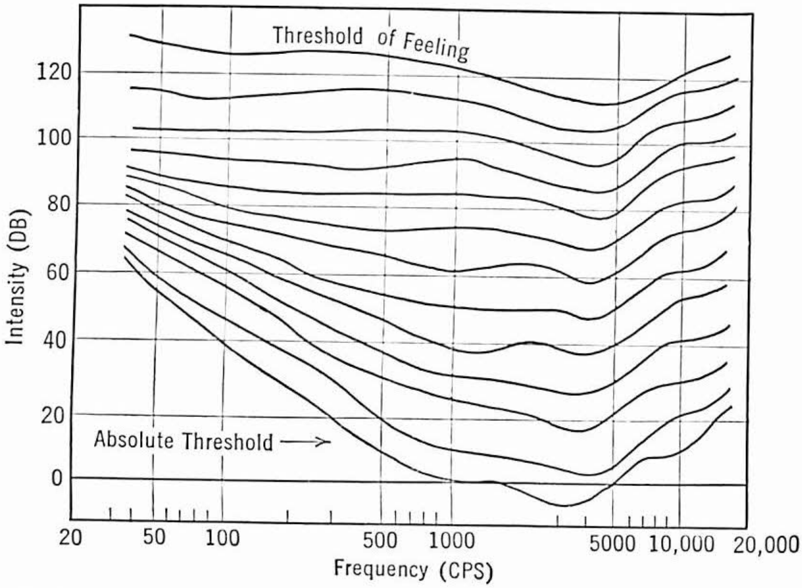


FIG. 11.13. "Equal loudness" contours. A sound falling at a given point along a line (contour) will be as loud as a sound falling at any other point along the same line. (From Stevens and Davis,[15] after Fletcher and Munson.)

Loudness: Terminal Threshold. The TL for loudness is actually a threshold for somesthesis from the ear structures. The uppermost line of Figure 11.12 is the TL. At this level of intensity, tickling, touch, and pricking are sensed in the ear. Above this level, additional intensity does not result in additional loudness. In a practical situation, if there is noise at or above the TL level, communication by sound is impossible — even if the voice were louder than the noise the extra intensity could not be discriminated. Noisy aircraft, the boiler factory, and some tanks approach or exceed this level.

Complex Sounds. It is not possible in a text on physiological psychology to consider in any detail the many aspects of psychoacoustics which have been explored in the interests of more efficient auditory communication, acoustical engineering, and related fields. Suffice it to point out that pitch and loudness thresholds are not the whole story. For example, the **harmonic** content of sound can be discriminated even when only a few percent of the sound energy is in the harmonics (overtones) . Much annoying distortion in sound-reproducing systems is in the form of harmonics generated within the equipment itself. In addition, sound engineers use a host of largely undefined terms to describe the characteristics of reproducing systems. There is no doubt that one can discriminate between " clean " and " muddy " reproduction, for example, and some of the physical characteristics underlying such judgments are known. The human auditory sense is capable of considerable qualitative discrimination in addition to simple pitch and loudness.

AUDITORY DEFECTS

Interference with hearing can occur at any point in the auditory mechanism. When the difficulty is at the cerebral cortical level, the effect is likely to be perceptual in nature. Below the cortical level, lesions in other CNS structures may lead to sensory difficulties. Our present concern will be with the peripheral mechanism.

Deafness. Deafness may be complete or partial. Partial deafness may result from numerous causes and the characteristics of the deafness will depend upon the causation. In the outer ear, accumulation of wax can lead to a considerable loss in sensitivity over the entire auditory range. This is a rather common cause of loss of sensitivity, but a very easily treated one. Infections of the outer ear, especially those affecting the drum, can lead to more serious loss of sensitivity. Ordinarily, such losses affect all frequencies rather evenly.

When the difficulty is in the middle ear transmission mechanism, the loss is most prominent for the lower frequencies. A common cause is the hardening of the junction of stapes and oval window (otosclerosis) so that normal transmission is interferred with. A hearing aid will be of considerable benefit in such cases.

When the inner ear is affected, loss of the higher frequencies first is most likely. This may result from several causes, the most common being ageing. Figure 11.14 shows the normal loss of sensitivity with increasing age. Another common cause in our noisy modern world is acoustical trauma, that is, the result of exposure to loud sounds. Since the results of traumata are important for auditory theory, they are discussed in Chapter 12. Inner ear defects sometimes result in what are called **tonal gaps** or **tonal islands** in the audible frequency range. A tonal gap is a region of loss surrounded by greater sensitivity. A tonal island is a region of sensitivity surrounded by loss. A common tonal gap occurs around 4096 cps, often after exposure to loud sound.

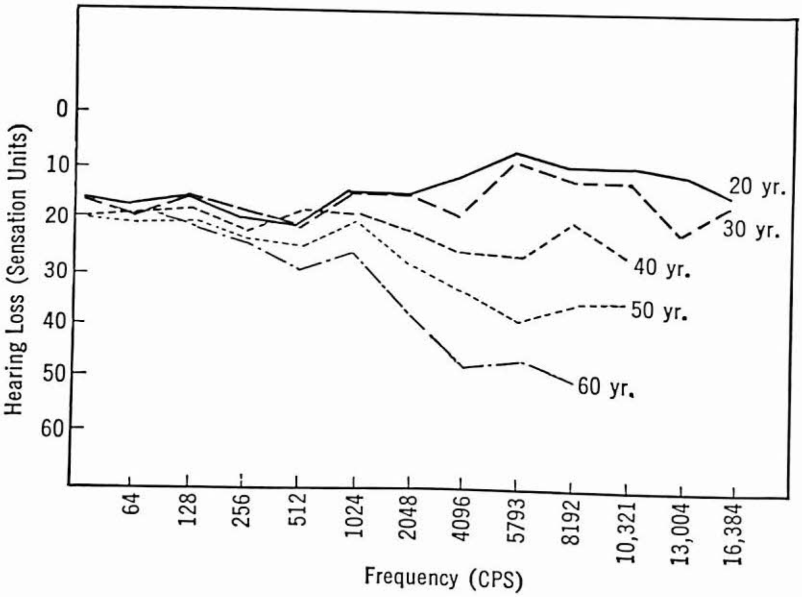


FIG. 11.14. Hearing loss as related to age. (From Bunch.[16])

An interesting phenomenon noted in cases of inner ear deafness is called **recruitment.** In such a case, the threshold for sound is very much raised, but after the abnormally high threshold is exceeded, the sound suddenly becomes very loud. The sufferer from this phenomenon often alternates between requesting others to speak more loudly and asking them to stop shouting. The exact cause is unknown, despite considerable speculation. A hearing aid is not to be recommended since, if the sound is amplified to a detectable level, it is heard as uncomfortably loud.

Complete deafness cannot result from difficulties in the transmission mechanism. If the inner ear and nerve structures are intact, the sound can be lead to the bones of the head through a suitable coupling device and stimulation of the inner ear will result. Severing of the auditory nerve, or complete destruction of the inner ear mechanism, as can occur from blast waves, will result in complete deafness. Occasionally persons are born without the cochleas, or even without inner ears at all. Such individuals are of necessity completely deaf.

Tinnitus. " Ringing ears," or **tinnitus,** is noticed occasionally by almost everyone. Sometimes it follows a loud sound, or accompanies a cold or sore throat. Some people have tinnitus continuously. Just as is the case with deafness, tinnitus may arise from defects any place in the auditory system. Often it is accompanied by dizziness. Many cases of tinnitus have resulted from exposure to loud sounds — e.g., aircraft, rifle fire, and riveting hammers. Such cases probably involve the inner ear. Still other cases can be relieved by improving the pressure equalizing function of the Eustachian tube. Perhaps the most amazing thing about tinnitus is that we don't all have it. Considering the extreme sensitivity of the ear, but little energy is needed to exceed the threshold.

Diplacusis. A disorder in which the same sound evokes different auditory sensations in the two ears of the same person is called **diplacusis.** A slight amount of diplacusis is normal. The two ears of a given person are never exactly alike. However, when the difference becomes great enough, it is most annoying. In severe cases, the sound heard by one ear may seem a half octave higher than that heard by the other. More commonly, the difference is about a half-semitone — enough to be very important clinically. The exact physiological explanation is obscure.

Other Binaural Effects. In addition to diplacusis, other aspects of **binaural** versus **monaural** stimulation require comment. First of all, the two ears of the same person are rarely of equal sensitivity. On the average, the two ears differ by about 6 db in absolute threshold — a very significant amount. If the two ears are stimulated simultaneously, the threshold is close to that of the more sensitive ear. If, however, we find a subject with ears which are equally sensitive, the binaural absolute threshold is 3 db less than that of either ear alone or, in other words, just half the power. It looks very much as though the threshold were in terms of power no matter whether applied to one or two ears. Half the power applied to each of two ears is the same as the whole amount applied to one ear. The bilateral connections of each ear are probably of importance in determining this outcome.

The loudness DL measured binaurally is also 3 db smaller than that measured monaurally. This result is best explained statistically.

Two ears have twice the neural elements of one ear; using both ears doubles the sampling of neural elements underlying discrimination. It happens — and this can be taken on faith for our purposes — that a 3-db decrease in DL is precisely what would be predicted from the statistical consideration of doubling the sample. In nonstatistical terms, using two ears increases the chances of detecting differences because more elements are involved. The summation of above threshold sounds presented binaurally is closely related. In general, sounds of the same frequency presented to the two ears at the same time add in loudness, so that, if of the same intensity, the resulting sound is just twice as loud as either monaural sound alone.

In the next chapter we shall be concerned with more complex effects of stimulation by more than one tone at a time and shall consider the evidence for the way in which the ear and auditory system accomplish their function.

Suggested Readings

1. Geldard, F. A., *The Human Senses.* New York: Wiley, 1953, Chaps. 6 and 7.
2. Chapanis, A., Garner, W. R., and Morgan, C. T., *Applied Experimental Psychology.* New York: Wiley, 1949, Chap. 7.

12 | *Audition: continued*

Cochlear Potentials. **Cochlear potentials,** or **microphonics,** are a very important facet of auditory response and their discovery and study has contributed immensely to our knowledge of the auditory mechanism. They properly belong in a general discussion of electrophysiological evidence but, since we shall find it convenient to refer to them several times before such evidence is considered, they are introduced here. Cochlear potentials are electrical responses generated within the Organ of Corti itself. These potentials are not action potentials and are non-nervous in origin. They reproduce very closely the form of the sound wave as shown in Figure 12.1. The magnitude of the electrical potential varies with the magnitude of the stimulus almost linearly up to fairly high levels. Figure 12.2 shows this function for a wide range of frequencies — it is a straight line during the lower part of its course, but departs from linearity at higher intensities, and finally flattens. Cochlear potentials induced by different frequencies have different spatial patterns on the basilar membrane. Low frequencies have a much greater spread, reaching the apex of the cochlea, whereas those induced by higher frequencies spread shorter and shorter distances. Figure 12.3 shows the relative response from the basal turn (nearest the round window), the second turn, and the apex of the cochlea of the guinea pig for a number of fre-

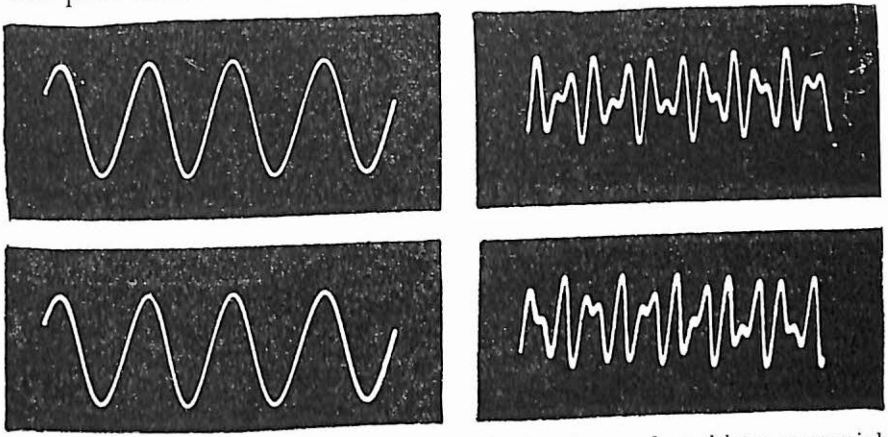


FIG. 12.1. Comparison of wave forms of stimulus and cochlear potentials for two tones — the stimulus waves are the top figures. (After Wever.[1])

quencies of stimulation. There is no response at the apex to tones of 1600 cps and above. At 10,000 cps there is no response from the second turn. Cochlear potentials are probably responsible for exciting the nerve impulse.[4] Thus, their study leads us closer to the last phases of stimulation than we are able to reach in other senses.

Tonal Interaction. In Chapter 11 the response of the auditory mechanism to tones presented singly was discussed. When a single sine wave is distorted somewhat, it gives a series of tones whose frequencies are whole multiples of the fundamental frequency as given by the Fourier analysis. Thus, a distorted 1000-cycle wave produces a complex wave which contains, in addition to the fundamental frequency of 1000 cps, frequencies of twice, three times, four times, etc., the fundamental — i.e., frequencies of 2000, 3000, 4000, etc., cps. The first few of these overtones are loud enough to be heard by a trained person. Such overtones are generated by physical objects, as we have seen in Chapter 11, but they are also generated within the auditory mechanism itself and become more prominent as the intensity increases. In the latter case they are called **aural harmonics.** Some of the things we hear are thus not present in the physical sound.

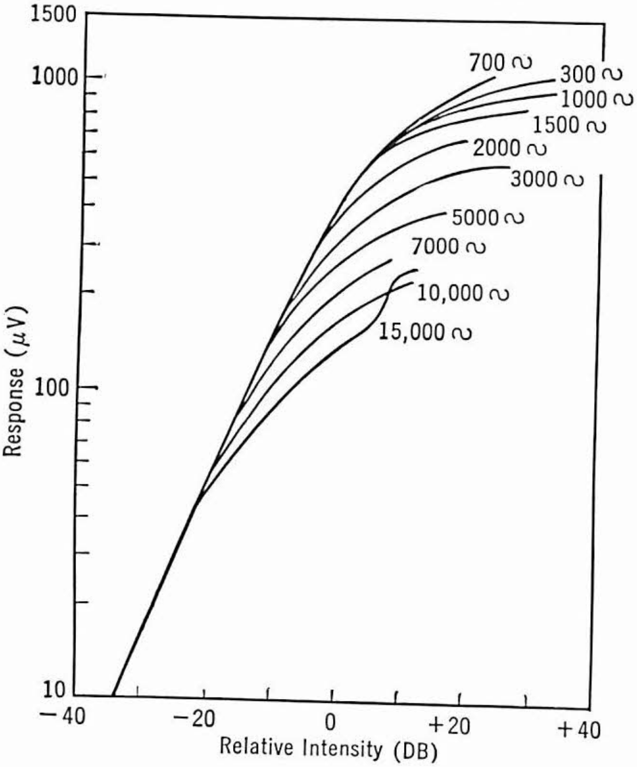


FIG. 12.2 Electrical potentials in microvolts at increasing intensities for tones of various frequencies (adjusted for differences in sensitivity). The distortion starts at lower intensities for tones of higher frequencies. (After Wever and Lawrence.[2])

When two pure tones are sounded together a number of things may happen, depending upon the frequencies and intensities of the two. When one tone is more intense than the other the most prominent effect is the **masking** of one tone by the other, the exact amount depending upon the relationship between the two frequencies. This effect will be discussed in a subsequent section. When the two tones are of approximately equal intensity either **beats** or **combination tones** may occur, depending upon the frequency relationships of the two generating tones.

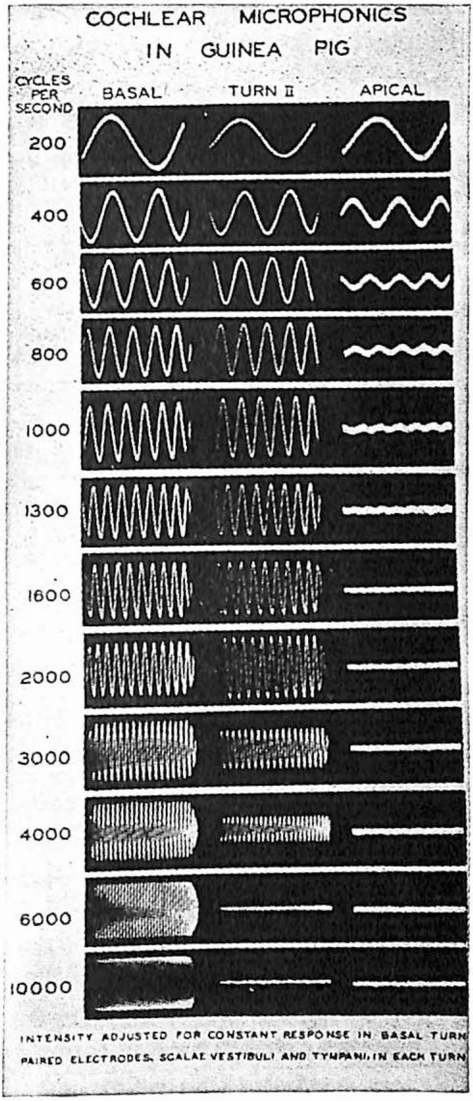


FIG. 12.3. Simultaneous oscillograms from Turns I, II, and IV of guinea pig cochlea for various frequencies of stimulus. The higher frequencies do not affect Turn III, and the intermediate and high frequencies do not affect the apex. (From Tasaki, Davis, and Legouix.[3])

Beats. When two pure tones of equal intensity and slightly different frequencies are sounded together, one hears a single tone which waxes and wanes in loudness as the two waves go in and out of phase. The fluctuations occur at a rate equal to the difference between the frequencies of the two original tones. If the tones are 1000 and 1004 cps, then one hears a tone which increases and decreases in loudness four times a second. There are three stages through which beats pass as the difference in frequency is increased, the exact limits of each depending upon the frequency of the standard and the intensity level. At first, with small differences in frequency, there is the waxing and waning of a single tone, as described above. At about 6 cps difference, the beat effect experienced is one of intermittence, which is unpleasant. At still higher frequency differences, say about 30 cps, the intermittence becomes a roughness and this may persist up to several hundred cps. The lower limit for beats is variable, but may be of the order of one beat in a minute or more. At intermediate frequencies an intertone can be heard, along with the two primaries, and it is the intertone which beats. Beats are utilized by piano tuners to obtain more exact tuning than can be reached by dependence upon pitch discrimination. They are also a very useful experimental tool in exploring for aural harmonics.

Combination Tones. At appropriate differences in frequencies of the two primary tones, combination tones appear as aural harmonies.

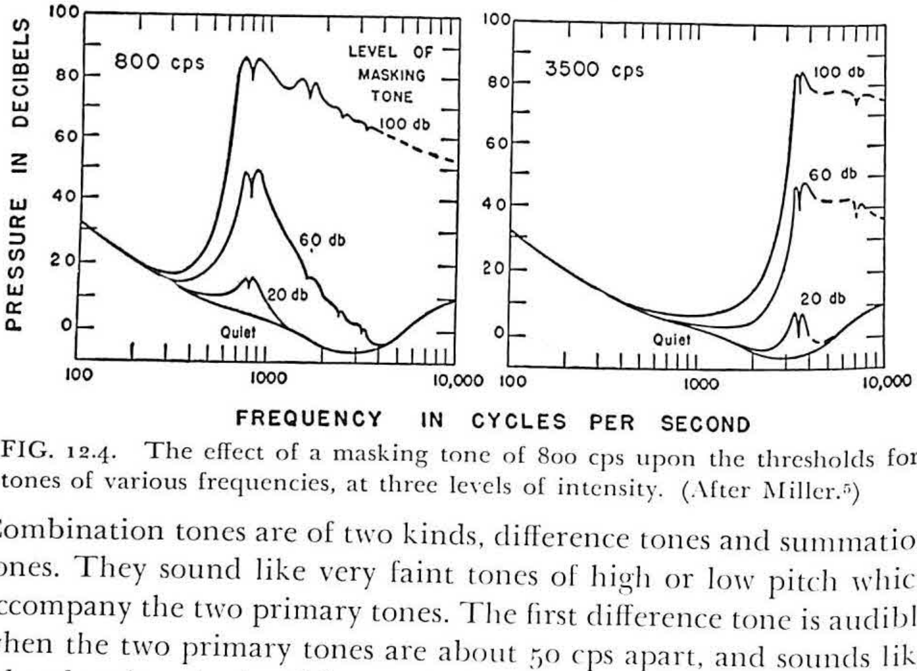


FIG. 12.4. The effect of a masking tone of 800 cps upon the thresholds for tones of various frequencies, at three levels of intensity. (After Miller.[5])

Combination tones are of two kinds, difference tones and summation tones. They sound like very faint tones of high or low pitch which accompany the two primary tones. The first difference tone is audible when the two primary tones are about 50 cps apart, and sounds like a low buzzing. As the difference in frequency of the two primaries is increased, the pitch of the difference tone rises. The first summation tone is much harder to hear; it has the same pitch as a tone of the frequency of the sum of the two primaries and, therefore, sounds high.

There are combination tones corresponding to all the sums and differences of the two primary tones, and all their whole multiples (the harmonics) in all combinations, as can be shown by recording the cochlear potentials. But, as with overtones in musical instruments, certain ones are emphasized. Only a very few of the combination tones, usually the first difference tone and the first summation tone, can readily be heard even by an experienced observer. The distortion which produces these aural harmonics does not arise from the middle ear, as was previously thought, but in the inner ear itself and, probably, in the actual receptor organs.

Masking. When the two tones presented to the ear are unequal in intensity, the more intense one tends to mask the other, the presence and strength of the effect depending upon the frequency relations between the two as well as their intensity relationships. The masking effect of a tone is measured in terms of the increase in threshold of the masked tone above its normal threshold. The masking effect is greatest in the frequency region of the masking tone. A given tone has the greatest masking effect upon similar tones, but two identical tones will summate. If beats are induced, masking fails. As the intensity of a masking tone is increased the band of affected frequencies spreads, but the spread is largely toward tones of greater frequency. Figure 12.4 shows the effect of a masking tone of 800 cps at three different levels of intensity. Note that the largest threshold increase is in the immediate neighborhood of 800 cycles for all intensities. The subsidiary bumps in the curves represent an increase in masking effect for multiples of the masking frequency. At high intensity levels, the 800-cycle tone shows a large masking effect upon all higher frequencies but very little upon those below it. In general, we may say that high tones do not mask low tones, but low tones mask high ones very effectively. The masking phenomenon probably arises in the cochlear nerve fibers: it does not appear in the cochlear potentials and is, therefore, not due to the receptor processes themselves.

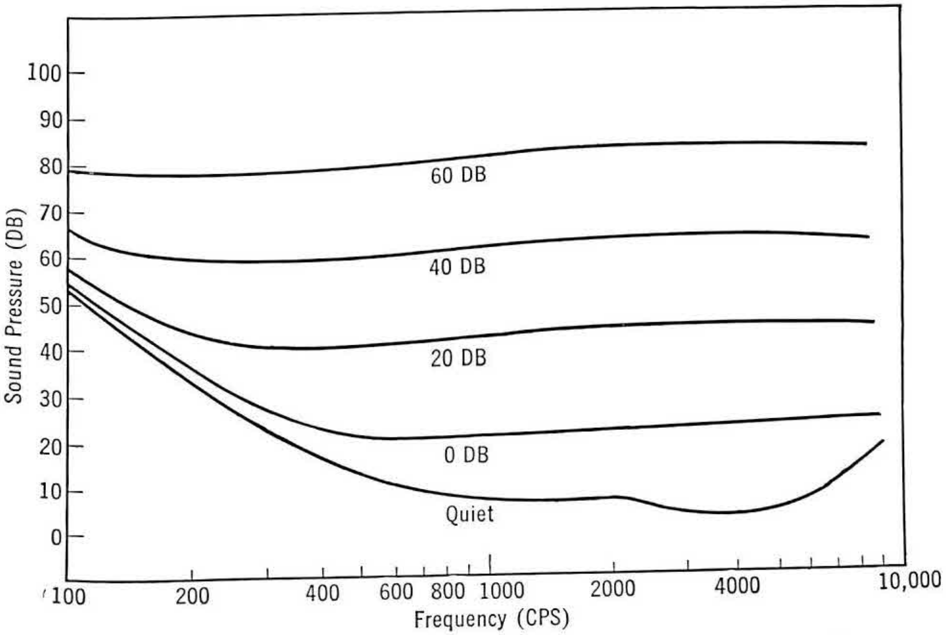


FIG. 12.5. The masking effect of white noise of four intensity levels upon tones of various frequencies. (After Hawkins and Stevens.[6])

A situation of some practical importance arises when we have masking of tones by noise. In any nonexperimental situation a significant level of background noise exists and this tends to mask music, speech, and other forms of signals. Since noise contains many frequencies, tones of many frequencies will be masked. Experimentally, "white" noise, noise containing all frequencies in equal amounts (by analogy with white light), is often used. Figure 12.5 shows the effect of white noise in raising thresholds for tones of various frequencies. White noise at 30 db masks only the intermediate frequencies, but at higher levels the masking effect is uniform throughout the frequency range tested. This means that there are no frequencies available which can be used for signals without increasing the intensity of the signaling tone so that it once more is brought above threshold. Noise levels of 90 db and above are not uncommon around machinery, and in submarines and airplanes, and the problem of communication between men in such situations is serious. Since the terminal threshold for audition is about 140 decibels, the usable auditory range is very much restricted under such circumstances.

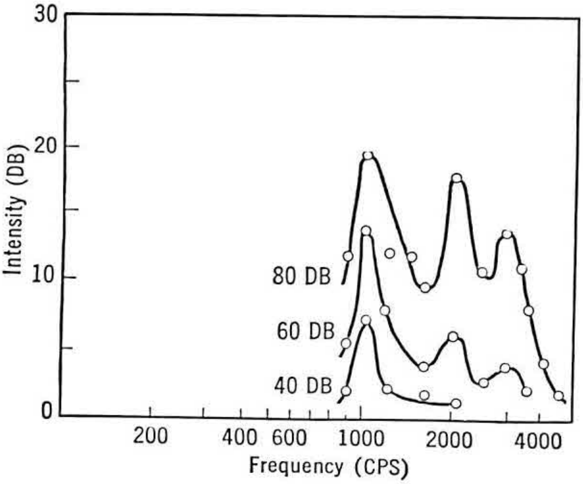


FIG. 12.6. Spread of adaptation for a stimulating tone of 1000 cycles, for 40, 60, and 80 db levels. (After Lüscher and Zwislocki.[7])

Adaptation. Auditory adaptation is not obvious to casual observation. The roar of traffic or the ticking of a clock can be heard at any time of the day, if one turns one's attention to it. It does not seem less loud after a period of time than it did at first; one merely ceases to attend to it. But adaptation, or, as it is more often called in audition, fatigue, is a very real phenomenon and can readily be demonstrated experimentally. The increase in threshold for a given tone can be measured after sounding another tone of a given frequency, intensity, and duration, and this increase is taken as the index of the fatigue effect. The frequency is important in two ways. First, the stimulating frequency has the greatest fatigue effect upon test tones of the same or neighboring frequencies. This effect can be seen in Figure 12.6, where the greatest hearing loss is for test tones very close to 1000 cycles. As with masking, there are subsidiary peaks at the harmonics of the stimulating tone. Second, there is a difference in effect between high and low tones. Below 600 cycles, and with tones of weak or moderate intensities (below 40 db), no fatigue is demonstrable, whereas it is significant for higher frequencies.[8] At higher intensity levels, the higher frequencies show a somewhat greater fatiguing effect.

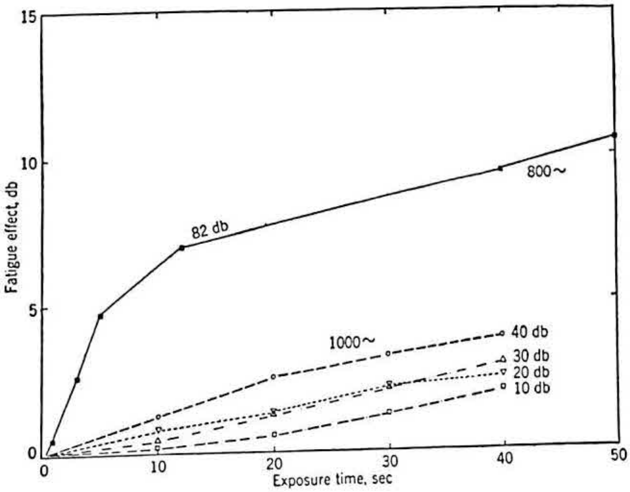


FIG. 12.7. Fatigue effect as a function of exposure time. (After Wever.[1])

The duration of the stimulating tone is another factor of importance. Even very brief tones increase the threshold of the test tone. Stimulating tones of less than half a second's duration are often used experimentally. Figure 12.7 shows the effect of increasing the exposure time upon tones of several intensity levels. In all cases there is an increase in fatigue as the duration increases. With high intensity the effect is even more pronounced. Figure 12.10 shows this effect for tones of extremely high intensity where the increase in fatigue is rapid with increasing duration.

Intensity of stimulation is also directly related to fatigue effect. This is obvious in Figures 12.6, 12.7, and 12.8 where increasing the intensity of the stimulating tone increases the hearing loss. Figure 12.6 also shows another phenomenon — the increasing spread of effect with increasing intensity. As the intensity is increased a greater range of frequencies is affected, but this spread is largely to frequencies higher than that of the stimulating tone. In this respect, also, fatigue is similar to masking.

Recovery from auditory fatigue is ordinarily very rapid. Figure 12.9 shows the recovery from fatigue for four levels of intensity as a function of time. Recovery is virtually complete for all reasonable levels of stimulation in under half a second. With very weak tones (5 db), recovery time is of the order of 20 milliseconds. Recovery time is increased by increasing both the intensity and the duration of the stimulation. In situations where men are exposed to high-intensity noise for extended periods of time, the reduction in sensitivity may be significant. In one study, exposure to submarine engine noise at the 110-db level produced sufficient hearing losses in all subjects to render speech communication less efficient than normal for a half hour or more.[11]

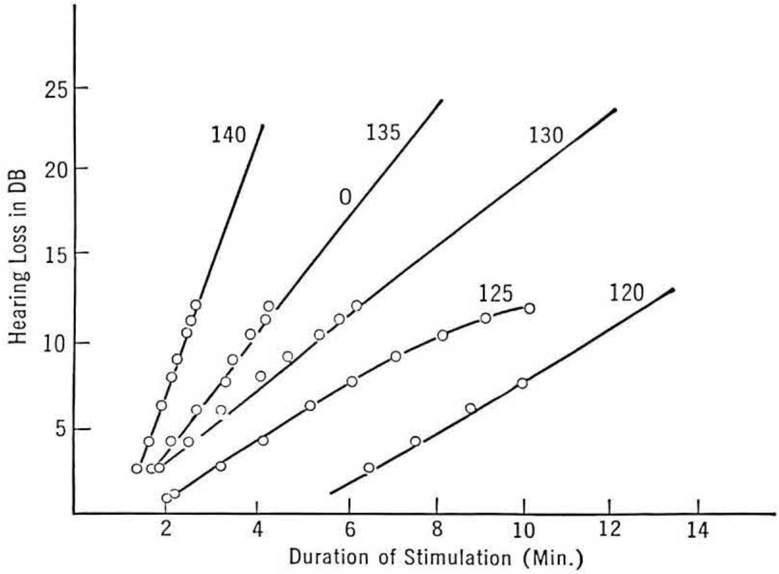


FIG. 12.8. Effect of duration of stimulation upon hearing loss for various intensities of stimulation with 750 cps tone, measured for 1000 cps tone 2 minutes after start of recovery. (After Harris.[9])

The locus of this fatigue effect is of some interest. Since it is a monaural process, that is, cannot be produced in one ear by stimulating the other, it is not a central process.[7] Neither is it to be found in the cochlear response. It is, therefore, judged to be a peripheral process, not in the receptor, but probably in one of the early neural processes. The fact that a fatigued ear shows loudness recruitment, as one finds in nerve deafness, is taken as additional evidence for this conclusion.[12]

There is no true after-image in audition. The after-effect of stimulation is not hearing some kind of a sound; there is no persistence of sensation after the removal of the stimulus. This is another example of the efficiency of the ear as an analytical instrument. Under very special circumstances there is a change in the quality of sounds, but the conditions under which this occurs are extremely artificial.

Stimulation Deafness. The step beyond fatigue in auditory stimulation is a real impairment in function which may be either temporary or permanent. The experimental work is done largely with guinea pigs because their auditory mechanisms are relatively more accessible, but the results are considered to hold reasonably closely for man because of similarities in structure. In these experiments, a tone of given frequency and great intensity is sounded for a period of time and the loss in electrical response of the cochlea (cochlear potentials) is measured. Later, a microscopic examination of the cochlea may be made to ascertain the extent and location of the damage to the structure itself. The reduction in response to low tones is greatest toward the apex of the basilar membrane, that to high tones is greatest near the basal end of the cochlea. Where the loss in cochlear response was 10 db, microscopic examination showed a predominance of abnormal hair cells in the corresponding part of the cochlea; where the loss was 30 db, there was severe damage to hair cells or supporting structures or both. Permanent injury is produced by a one-minute exposure to a tone of greater than 140 db.[13] Figure 12.10 shows the results of microscopic examination of the cochlea after stimulation damage by tones of four different frequencies. The top figure shows extensive damage at the apical end of the basilar membrane and an average loss (as measured by reduction in cochlear

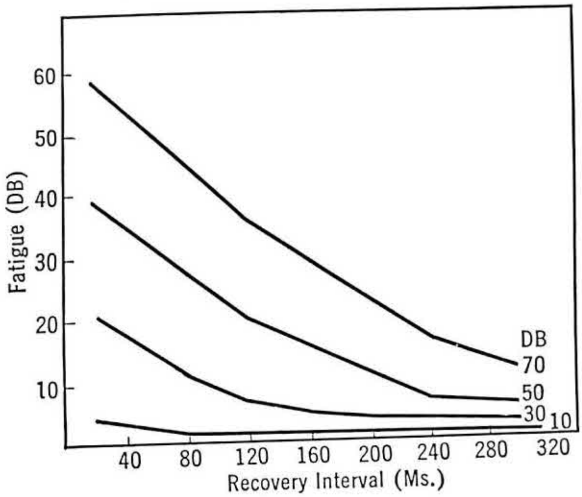


FIG. 12.9. Recovery from auditory fatigue induced by a 3000 cps tone applied for .4 second. (After Rawnsley and Harris.[10])

potentials) of 39 db. As the frequency rises, the damage shifts toward the basal end, but also becomes much more restricted in area, and, in these cases, the average loss in cochlear response is less for the higher frequencies.

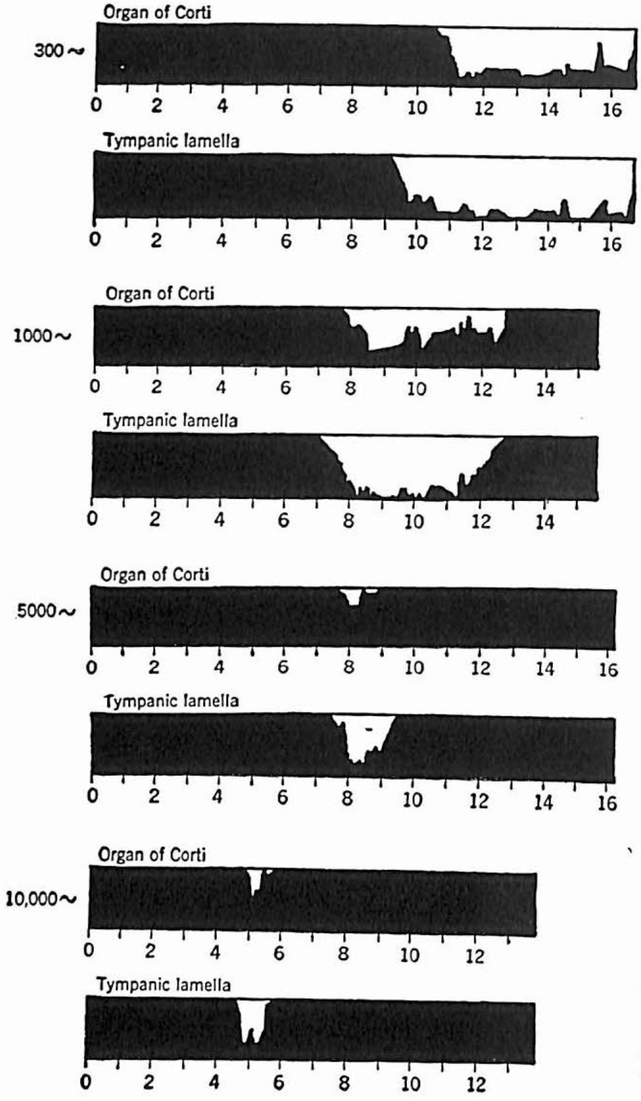


FIG. 12.10. Lesions produced in organ of Corti by tones of various frequencies at 1000 dynes per cm^2 for 4 minutes. Black areas of full height denote no microscopic damage, various proportions of white indicate various proportions of damage at the indicated distance from the basal end of the basilar membrane. (Wever[1] after Smith.[14] and Smith and Wever.[15])

Where people are subjected to intense sounds over long periods of time, or to very intense sounds momentarily (as in an explosion close at hand), permanent damage may result. There are large individual differences in susceptibility in man, as in guinea pigs. A common defect is the " boilermaker's gap " at about 4096 cps, but often the reduction in sensitivity is a more general one. Tinnitus may also result from overstimulation. In experimental work with human beings at intensity levels producing temporary hearing losses.

there is some evidence that high-frequency response is most affected.[16] Figure 12.11 shows the hearing loss induced by a 1000-cycle tone at 130 db at four different durations. Note the spread of effect upwards toward the higher frequencies as with masking and fatigue. This hearing loss was of a temporary nature.

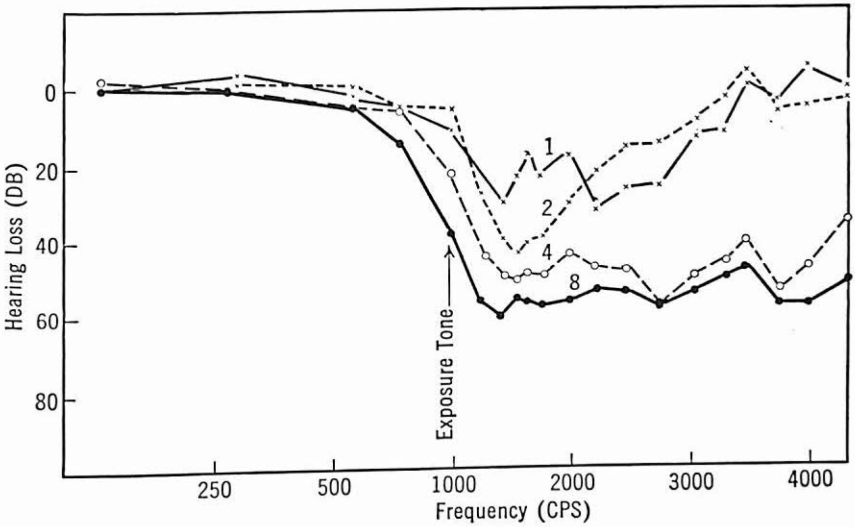


FIG. 12.11. Hearing loss induced by a 1000 cps tone at 130 db for 1, 2, 4, and 8 minutes' exposure. (After Davis, Morgan, Hawkins, et al.[16])

Localization of Sound. The localization of sound involves complex sensory and perceptual processes. In the simplified experimental situation using pure tones, there are two factors of major importance: phase differences and intensity differences at the two ears. The efficacy of these two factors depends upon the frequency of the sound wave, phase differences being most useful at low frequencies and intensity differences at high frequencies. Figure 12.12 shows the variation in error of localization as a function of frequency — errors

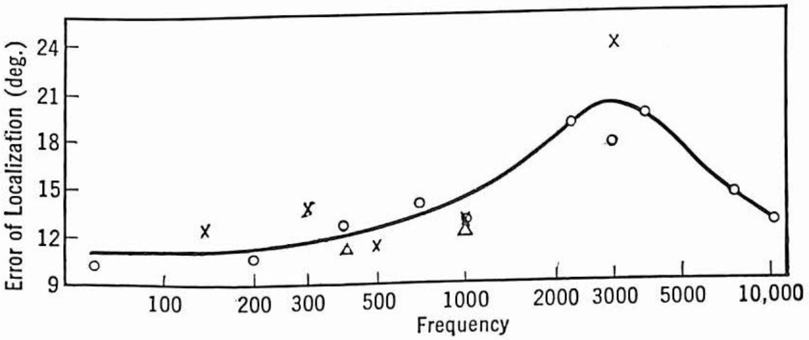


FIG. 12.12. Errors of localization as a function of frequency. (After Stevens and Newman.[17])

are relatively small at low frequencies, but gradually increase up to about 3000 cps and then decrease again above 4000 cps. There is evidence that observers can detect differences in phase of two tones led to different ears up to about 1500 cps but not for higher frequencies. There is also evidence that differences in intensity led to different ears can be detected with greater accuracy for high than for low tones. But such a statement oversimplifies the situation, for there are rather large differences in accuracy of localization under different conditions — room, sound cage, or open air; pure tones, complex tones, or noises. Actual measurements of the differences in sound pressure at the two ear drums have shown a significant sound shadow cast by the head, which is greatest when the source of the sound is to one side. The effect is most marked for high frequencies because sound waves of long wavelength (low frequency) can bend around corners more sharply than those of short wavelength (and high frequency). There is also an appreciable shadow cast by the pinna which is effective for the discrimination of front from back.[18] Complex sounds, like clicks and hisses, are more readily localized than pure tones, for all cues may be used, since both high and low frequencies are present.

In normal life, localization cues are not confined to purely auditory ones. Visual cues are used and often predominate over auditory ones, a bell-like sound being perceived as coming from a seen telephone instead of from a hidden source. Head movements enable one to vary the sound at the two ears and to maximize the cues, not only for right-left discriminations, but also for up-down positions, which do not have other cues and for which localization is otherwise poor. Learning must play a role in the development of accuracy, integrating the above cues in the perceptual process and enabling the organism to compensate for the fact that there are binaural differences in perceived loudness of a sound which may amount to as much as 25 db.[19] In an ordinary room, where sounds are reflected from a great many surfaces, one would think that localization would break down, yet we know that we can locate a sound rather accurately under such circumstances. Some of these waves are, of course, much weaker than others, but of those which are nearly equal in loudness brief ones in close succession are fused and the localization is determined by the first of the two.

Speech. The topics of speech and communication are broad ones with many ramifications into which we cannot go, but there are some aspects which are interesting in connection with the information we have been discussing. The frequency-intensity plots of normal speech under average circumstances may serve to make some of the preceding discussions more meaningful. Figure 12.13 shows such a distribution for average conversational speech for men and for women. Most of the energy is at low frequencies for both sexes, below 1000 cps, in fact, but these frequencies are not the most important for intelligibility of speech. It should be noted that the curves for men and women are remarkably similar, except that the masculine curve shows a slight increase around 300 cps, while the feminine curve shows a slight increase above 5000 cps. The average intensity levels of these curves are somewhat above 70 db at 30 centimeters from the speaker, for average conversation. About 1900 cps is the midpoint in the band speech frequencies. If all frequencies above this are eliminated only 50 percent of test material heard can be recorded correctly and, similarly, if all frequencies below 1900 cps are eliminated. Elimination of the high frequencies affects the consonants more than the vowels and elimination of the low frequencies is more effective in reducing the intelligibility of vowels.

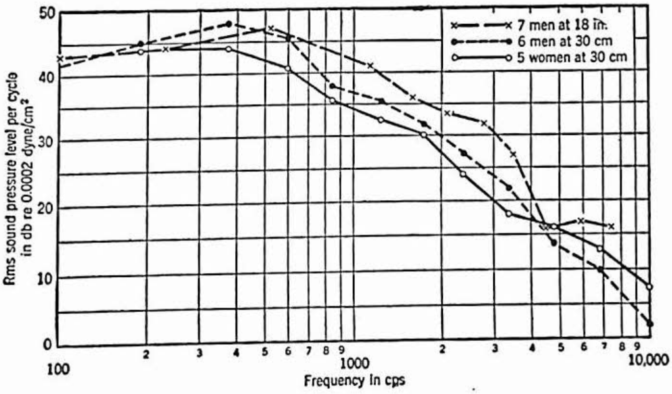


FIG. 12.13. Intensity of different frequencies as found in normal speech. Over-all level at 30 cm. from speaker; men, 76 db, women, 73 db. (From Licklider and Miller.[20])

Masking of speech by other sounds results in lower intelligibility due to missed syllables, although speech is rather resistant to interference because of great redundancy and the amazing ability of human beings to fill in a very sketchy sensory pattern perceptually. It is found that low frequencies produce greater masking than do high ones (cf. discussion of masking and Fig. 12.6).

The effect of frequency distortion is important in the design of radios, telephones, and the like. While it is possible technically to design and build electronic equipment which will reproduce sound with great fidelity (so-called "high fidelity" music systems are concerned with this problem), it is very costly and for most purposes unnecessary. For speech, the frequency range of 200 to 10,000 cycles is used and intelligibility drops as one progressively restricts the band width. If the band width is restricted to 600–4000, then the intensity level must be raised 10 db in order to achieve even a 60-percent score on a test of intelligibility in the presence of noise. For music, the restriction of band width is more serious. Listener tests, comparing various restricted ranges with the full-frequency range (usually 30 to 15,000 cps in good sound systems), show a noticeable deterioration in musical quality with any restriction whatever, though, if any distortion is present in the full range, a somewhat restricted but distortionless reproduction is preferred. For this reason many manufacturers of radio equipment use a restricted range of frequencies, since increasing frequency range and maintaining quality results in sharply increasing costs.

Electrophysiological Evidence. Recording of action potentials has now been done at all levels of the auditory system, from the short, almost inaccessible primary neurons to the cortex itself. The characteristics of the potentials in various locations differ in form of wave, amplitude, latency, and frequency, as well as in responses to more complex aspects of stimulation. The action potentials of the auditory nerve show characteristics quite different from the cochlear microphonics previously discussed and quite similar to other sorts of action potentials. One of the major differences is the inability of the single fiber or even of the whole auditory nerve to " follow " the frequency of the stimulating sound wave. The cochlear microphonics follow very closely but action potentials do not. In the single fiber of a primary neuron in the auditory nerve, the upper limit for synchronization of the response and the stimulus appears to be about 400 impulses per second, although even this rate is not maintained for long. This means that an impulse appears at the crest of every sound wave in the cochlear potential, which always follows closely the stimulus wave (cf. Fig. 12.1). At the critical point, the nerve fiber can no longer follow and its rate of discharge drops suddenly to half. The fiber is then discharging at the crest of every second wave in the cochlear potential. At still higher stimulus frequencies, the fiber will discharge at the crest of every third or fourth wave. At about 2000 cps, the synchronization in the single fiber is no longer apparent but, if one records from many fibers in the whole auditory nerve, it is apparent that there is orderly discharge within the total group, for synchronization is apparent up to about 4000 cps.

As one traces impulses higher and higher in the central auditory pathway, the rate at which synchronous responses occurs decreases. In the medulla, a synchronization has been observed up to about 2500 cps, at the inferior colliculi, up to 1000 or possibly 1500 cps, and still lower in the cortical projection fibers. In the cortex itself, synchronization is not evident. Characteristics of action potentials at various levels of the auditory system are shown in Figure 12.14. Note that there is a progressive change in characteristics as one goes from lower to higher levels (bottom to top). The wave form becomes progressively smoother; the latency, duration, and amplitude of the response increase. Greater intensity of stimulus usually results in more impulses per unit time.

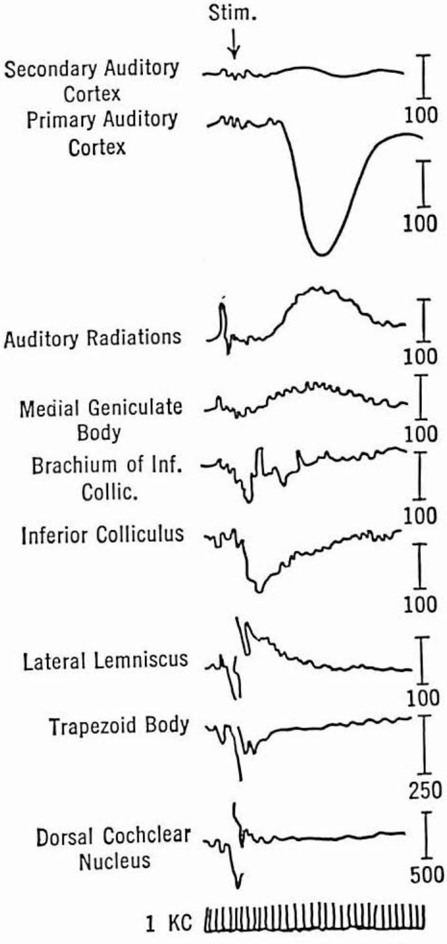


FIG. 12.14. Oscillograms from nine stations in the central auditory pathway. (After Ades and Brookhart.[21])

At all levels there appear to be fibers which are most sensitive to a restricted range of frequencies. This "tuning" is relatively sharp for threshold intensities, but broadens as the intensity is increased. Figure 12.15 shows the frequency-intensity response curves for four different secondary neurons. Note that the high-frequency slope is virtually a straight line, that is, the cut-off is very sharp at the high-frequency end. Figure 12.17 shows a similar response curve for a recording site in the cortex; the spread of response increases as the intensity increases, and the spread is almost entirely toward the lower tones.

There is **topographical organization** present to some extent at all levels of the auditory system; that is, the place of origin in the cochlea is reflected in the spatial arrangement of neurons. Recording of responses from single nerve fibers of the primary neurons has shown that those fibers arising from the basal turn of the cochlea may respond to tones of any audible frequency, but those arising from the apical end respond only to tones of low frequency. There is a somewhat more obvious spatial arrangement of neurons responding to tones of different frequencies in the inferior colliculi and in the medial geniculate bodies. The spatial localization in the cortex is extremely sharp. Figure 12.16 shows the spatial organization of frequency representation in the cortex of the dog. In this study it was found that the frequency shifted one octave for each 2-millimeter displacement in the anterior-posterior direction (for intermediate frequencies) and could even be shown to shift

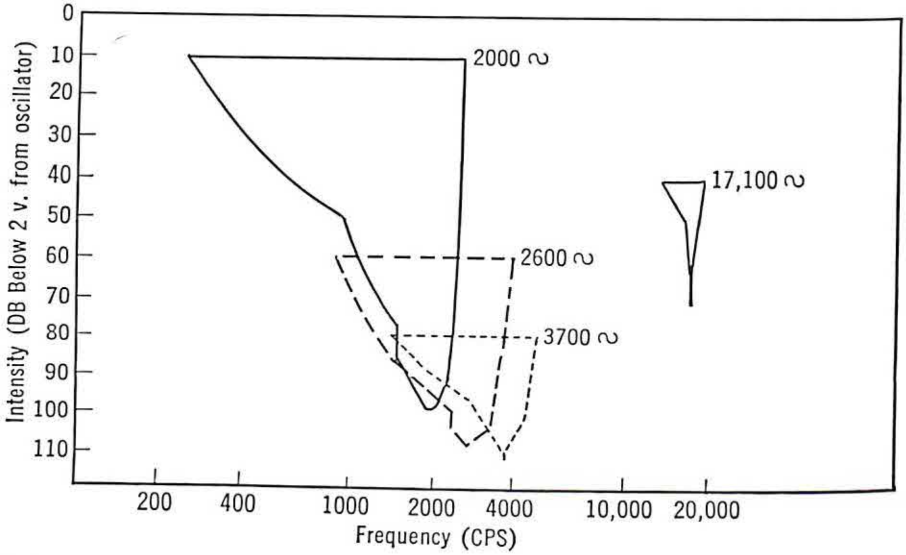


FIG. 12.15. Response areas for four different secondary neurons. (After Galambos and Davis.[22])

.1 octave per .2 millimeter displacement. This sharpness of position is limited to threshold levels of intensity, however; the higher the intensity the wider the band of frequencies to which a given patch of cortex will respond, as is evident in Figure 12.17.

Auditory Theory. Current auditory theories are much too complicated and require too much knowledge of physical principles and of neurophysiology to present here in any detail. But an attempt to

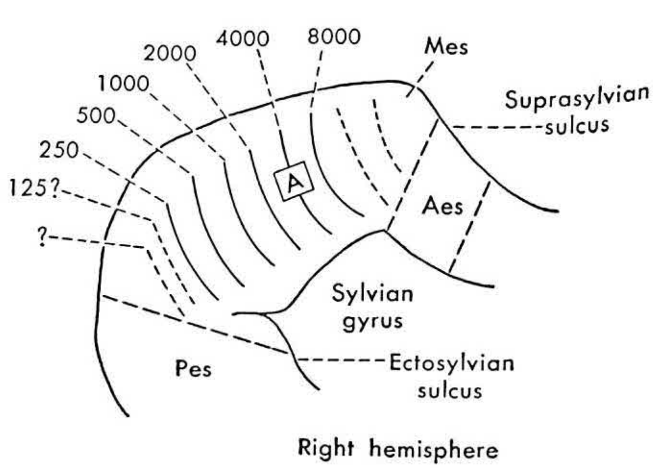


FIG. 12.16. Bands of cortex in middle ectosylvian gyrus of dog which responded to stimuli of various frequencies at threshold intensities after strychninization. (After Tunturi.[23])

present a simplified version of a combination of theories will perhaps serve to draw together a number of facts. As in other areas, different theories arise because of different emphases in research, some authors turning their attention primarily to one phase of this extremely complicated system and some to another.

It is useful to consider the auditory system as a two-stage system. In the first stage, there is a mechanical analysis by the cochlear structures; in the second, there is neural analysis by all the parts of the nervous system involved in hearing. The mechanical analysis stems from the purely mechanical properties of the cochlea, which are not only complicated but involve such minute structures and movements that research is rendered difficult. Recent experimental evidence has led to the conclusion that the basiliar membrane is not under tension, so that it does not vibrate as a piano string does. It does change in width significantly, being rather narrow at the stapes end (basal turn) and relatively wide at the apex. The vibration of the stapes sets up a traveling wave in the cochlea which produces a region of maximum displacement at some particular point. The region of maximum displacement depends upon the frequency of the sound wave; it is further from the stapes for low tones (which affect the apical end of the cochlea more strongly) and close to the stapes for high tones. Thus, there is a rough frequency analysis performed by

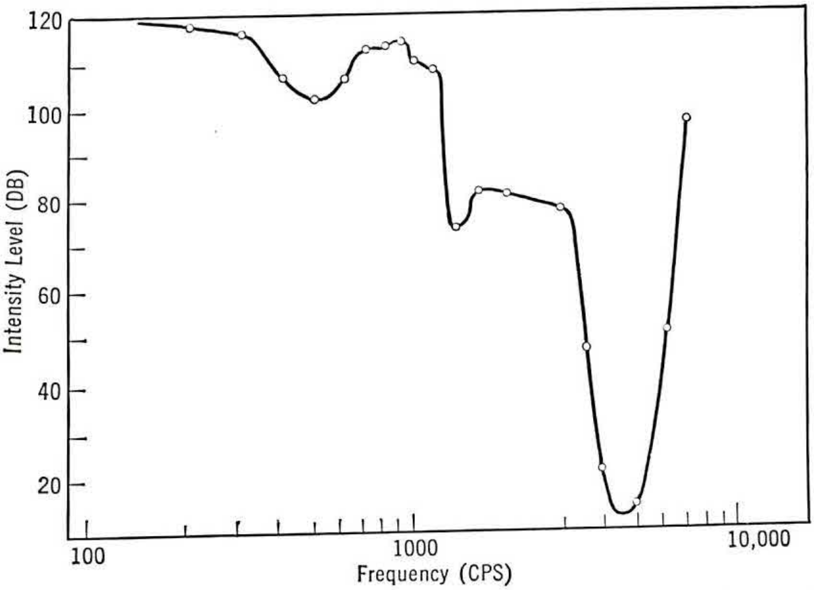


FIG. 12.17. Response curve of patch marked A in preceding figure. Area above the curve is the intensity-frequency range in which all tones are effective. (From Tunturi.[23])

the cochlea itself. Presumably, this maximum displacement produces in turn a maximum bending of the hair cells in the Organ of Corti which rests on the basilar membrane. This bending then is responsible for a small flow of electric current, which is picked up by recording devices as the cochlear microphonic and which excites the nerve impulse. This frequency analysis by the mechanical properties of the cochlear system is rough, not nearly acute enough to account for the accuracy of pitch discrimination, and it is less acute for loud tones than for near-threshold tones. Loudness is presumed to depend upon the number of nerve fibers excited. This in turn would depend upon the spread of displacement to a broader region and the excitation of less sensitive fibers in the region originally subject to the maximum displacement at the particular frequency.

The neural system is somehow responsible for the sharpening in frequency analysis which must occur between the mechanical analysis and the perception of pitch. Here the problem is like that which arises in visual acuity, where one also has a relatively gross analysis by receptors and accessory mechanisms, and the factors responsible are probably in part similar. These factors are not well worked out as yet, but probably involve higher order neurons with differing properties, such as elements which respond when a stimulus is removed, and inhibitory elements of various types. The frequency analysis of the cortex in terms of position is very sharp and this sharpness appears to develop gradually as one ascends to higher levels in the system. Hand in hand with this, the synchronization of impulses decreases as one ascends to higher levels. Thus, place analysis and frequency in the nerve fiber group both play a part, and possibly at some station in the system there is a translation from one to the other. It may be useful to consider the auditory system as a modified touch sense. They both respond to mechanical deformation of hairs. In hearing, there are accessory structures which heighten the sensitivity greatly, but the basic sensory change is similar. As in touch, identification of place stimulated does not depend upon the excitation of a single spot. In both cases there are many fibers and branches involved and the accuracy of localization stems from what the central nervous system is able to do with the pattern of signals which it receives. The central nervous system is not a placid lump of protoplasm, but a complex and dynamic system, and it should not be surprising that it makes a significant contribution to the acuity of perceptions of all sorts.

Suggested Readings

1. Geldard, F. A., *The Human Senses.* New York: Wiley, 1953, Chaps. 6, 7, and 8.
2. Wever, E. G., *Theory of Hearing.* New York: Wiley, 1949.
3. Wever, E. G., and Lawrence, M., *Physiological Acoustics.* Princeton: Princeton University Press, 1954.

13 | *Vision*

The importance of vision as a sense need not be belabored. It appears, however, that it is considerably more difficult for the student to apply what he has learned about sensation to vision than in the case of any other sense. In particular, the principle that what is sensed is determined to a large extent by the characteristics of the organism seems harder to grasp. That color, for example, does not reside in the object, that a book is not *really*, ultimately red, even though most of us see it as red, is difficult to accept, in spite of the fact that the principles of the adequate stimulus and of adaptation are well known. Somehow one is better able to detach the odor from an object than its color or shape or position. Vision is such a large and complex field that it becomes necessary to choose one area and its relevant facts and to give scantier coverage to other aspects. In keeping with the purpose of this text, we will emphasize the psychophysiological aspects and will accord very brief mention to the physical and industrial approaches, important as these are in professional-level knowledge of the field of color today.

Anatomy and Physiology. The eye is a roughly spherical structure embedded in a deep bony socket. It is held in place and its movements are controlled by three pairs of striated muscles which are responsible for **convergence.** Figure 13.1 shows the gross structure of the eye.[1] Essentially it is a semielastic body, having three outer layers enclosing a liquid-filled interior. Its outer layers or coats are pierced by the optic nerve (cranial nerve II), which emerges on the nasal side of the midline of each eye. The three outer coats are, from outside in: the **sclerotic coat,** a tough protective membrane, the **chorioid,** a layer containing blood vessels (and continuing forward, the **iris**); and the **retina,** the layer containing the receptors themselves and many nerve cells and connecting fibers. At the front of the eye, the sclerotic coat becomes transparent and is called the **cornea.** The eye is divided into two major portions by the **lens** and its accessory structures. The cavity between the cornea and the lens is filled with a viscous, clear liquid called the **aqueous humor.** The cavity between the lens and the retina is filled with liquid having slightly different characteristics, called the **vitreous humor.** Light must thus pass through numerous layers before reaching the retina; it must pass through the cornea, the aqueous humor, the lens, and the vitreous humor, and between each of these it is refracted to some degree and, also, some scattering of light occurs. The greatest amount of refraction occurs at the cornea.

The lens is responsible for our ability to bring objects at various distances into clear focus. Changes in focus, i.e., **accommodation,** are brought about by means of the **ciliary muscles,** smooth muscles which allow the lens to flatten when they relax, and allow the lens to bulge when they contract. When the lens bulges, near objects are brought into focus. The iris contains various amounts of a brown pigment which gives color to one's "eyes" — light-colored eyes contain little or no pigment. The diameter of the opening in the iris — the **pupil** — is controlled by two sets of smooth muscles in the iris under the control of the autonomic nervous system. One of its important responses is to changes in illumination; it becomes very small (about 2 millimeters in diameter) in bright light, and becomes large (about 8 millimeters in diameter) in very dim light. The latter is one of the factors in dark adaptation.

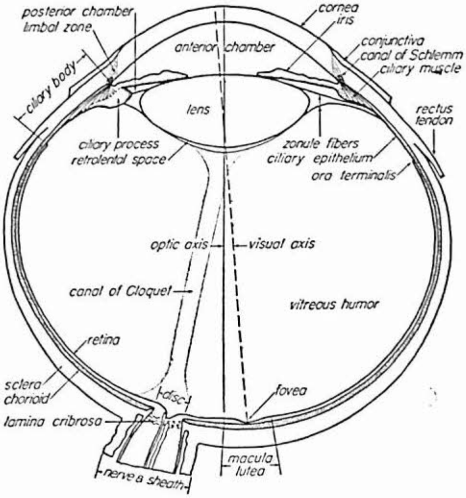


FIG. 13.1. Diagram of gross anatomy of the eye. (After Walls.[1])

The retina is a very complex structure. Figure 13.2 is a schematic diagram showing the ten layers which have been described.[2] The visual receptor cells, the **rods** and **cones,** lie in the outer layers of the retina. In the layers interior to them lie a number of kinds of **bipolar** and **ganglion cells** and large numbers of connecting nerve fibers. The greater part of the retina is thus seen to be taken up with nerve cells and their connections. One fact should be noted well — the nerve cells lie interior to the receptors and, thus the light must pass through the layers of nerve cells and fibers in order to reach the light-sensitive receptor cells. Awkward as this arrangement seems, it works remarkably well. At the region of clearest daytime vision, where the light rays are focused when one is looking directly at an object, some modification of the retina occurs. This region is called the **fovea** (see Fig. 13.1) and here the inner layers of nerve cells and fibers are pushed aside, making an indentation, so that the light has more ready access to the receptors.

The rods and cones have many points of similarity. They are elongated cells, with the cell bodies toward the inner ends of the cell, and with the photosensitive substance in the outer segments of the cell. Figure 13.2 shows the general structure of each. The rods are more cylindrical and thinner than the cones and the photosensitive pigments are different. These photosensitive pigments are transformed by light into slightly different chemical compounds. During this process energy is given up, which initiates further changes in the visual system. The chemical changes are now rather well understood in the case of the rod pigment, **rhodopsin**, but not as yet worked out for the cone pigment (or pigments), **iodopsin**. These changes in the photosensitive pigments are but the first step in a complex system.

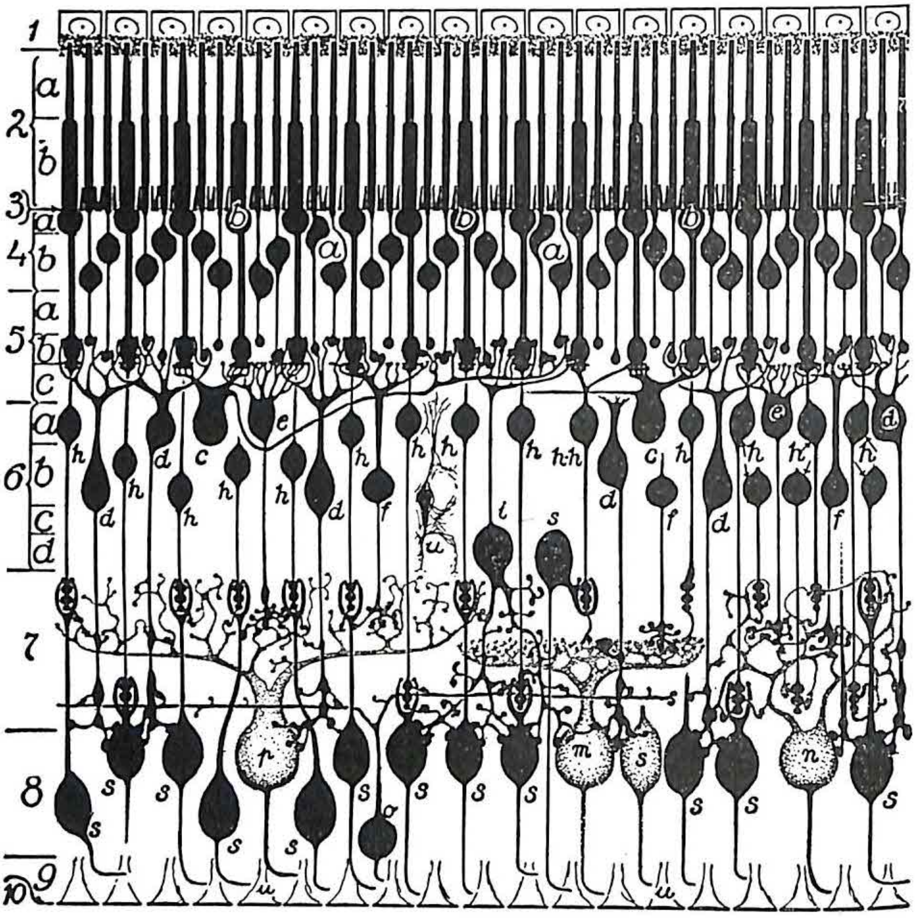


FIG. 13.2. Schematic diagram of retina. A, rods; B, cones; H, D, bipolar cells; S, ganglion cells. (After Polyak.[2])

The nature of the process which actually initiates the nerve impulse is as yet unknown.

The rods and cones have different distributions in the human retina. Only cones are found in the fovea, where they become very slender, are thickly packed, and have direct one-to-one relations with some bipolar and ganglion cells (one cone being connected with one bipolar cell which is in turn connected with one ganglion cell) as well as the usual diffuse relationships. This is the area of clearest daytime vision and of maximum color vision. The cones are thus the receptors for high illumination and for color vision. As one goes toward the periphery, the cones become fewer, until the extreme periphery is reached where there are few cones, diffusely connected. Contrariwise, the rods begin to appear outside the foveal region and increase in number as one goes toward the periphery, up to about 20° from the fovea. They are the receptors for night vision and, since there are no rods in the fovea, one must look a little to one side to see a dim object or a faint star; the fovea is blind in very dim light. The rods are color-blind, which is well-described in the saying "At night all cats are gray."

The middle layers of the retina contain the bipolar cells, nerve cells which are intermediaries between the rods and cones and the cells which provide the fibers for the optic nerve, the ganglion cells. The bipolar cells are connected to one or several cones, or to several rods, and are in turn connected with one or several ganglion cells. (There are also cells in the middle layers which interconnect parts of the retina, allowing retinal interaction.) The ganglion cells send their axons streaming across the retina toward the point of exit of the optic nerve. All fibers leaving for the central nervous system gather together at a single region and the eyeball is pierced only once. Since there are many fibers involved, the **optic disc,** as the region of exit of the optic nerve is called, is of appreciable size. It contains no receptors and is blind, hence the name, "blind spot." Images falling entirely within the blind spot (and not falling on the retina of the other eye) are not visible, but images which extend outside the blind area are filled in and one is not even aware of the existence of it without special techniques. The optic disc is about 15° toward the nose from the fovea and appears in the outside portion of the visual field for the given eye.

It is probably obvious by now that the visual system is quite different from other sensory systems neuroanatomically. Note that the first and second synapses of the system occur within the retina itself, so that the neurons which send their axons to form the optic nerve are third-order neurons. Figure 13.3 shows schematically the course of these fibers and the subsequent connections in the CNS.[2] Note that the axons from the right side of both retinae go to the right side of the brain because the fibers from the inner side of the left eye cross in the **optic chiasm.** Similarly the fibers from the left side of both eyes go to the left side of the brain. The optic nerve fibers have two major terminal stations. Some fibers terminate in the superior colliculi, where connections are made to the motor nuclei of cranial nerves III, IV, and VI, serving reflex eye movements. The other fibers, by far the largest number, travel to the lateral geniculate bodies in the thalamus, where they synapse with the cortical projection fibers. The latter go directly to the cortex of the occipital lobes (area 17), which is the primary sensory area for vision. Spatial relationships are well maintained both in the lateral geniculate body and in the cortex, although there are many more fibers in the cortical projections from the thalamus than in the optic nerve itself.

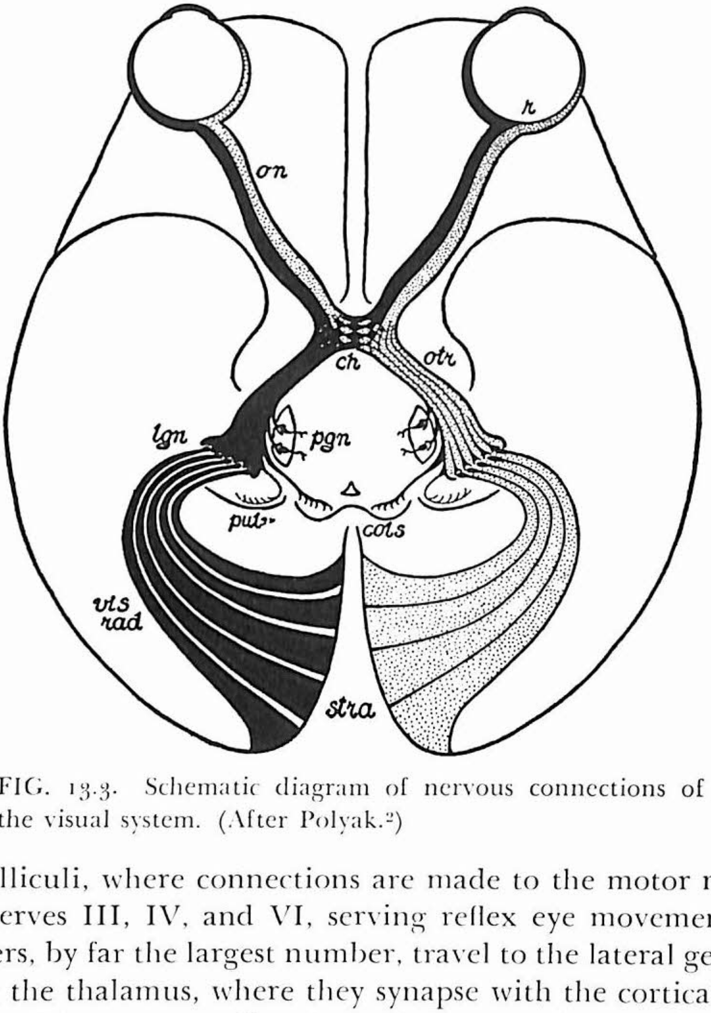


FIG. 13.3. Schematic diagram of nervous connections of the visual system. (After Polyak.[2])

The Stimulus. The adequate stimulus for vision is electromagnetic radiation of certain very restricted wavelengths. The entire band of electromagnetic radiation extends from very short wavelengths, where X-rays are found, through the ultraviolet to visible wavelengths (light waves), and then on through the infrared, or heat rays, to the long wavelengths associated with radio waves. Figure 13.4 shows the **electromagnetic spectrum.** The visible spectrum is seen to be but a very small portion of the total. The usable limits of the visible spectrum lie between about 400 **millimicrons** and about 750 millimicrons. Ordinarily wavelengths beyond these limits are not stimulating to the eye. The visual receptors can be stimulated inadequately by pressure and electric current. You can easily demonstrate the former to yourself by turning the eyes far to the right and pressing on the left eyeball at the extreme left-hand side with the finger tip. A small colored spot will appear in the extreme right visual field because of the mechanical stimulation of the receptors at the extreme left margin of the retina.

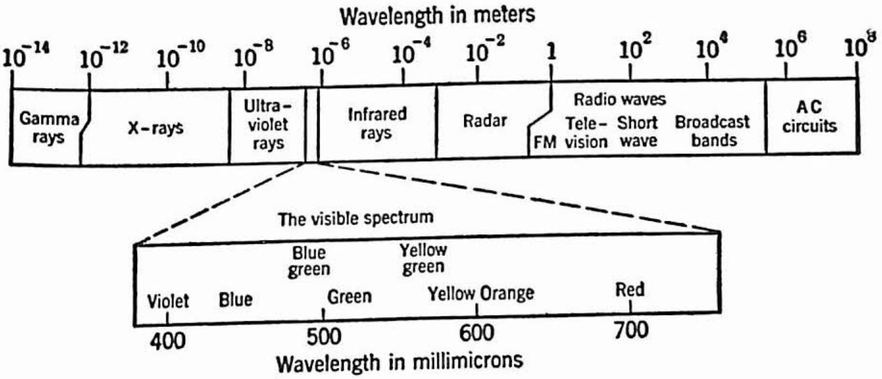


FIG. 13.4. Diagram of the electromagnetic spectrum showing the relative size of the visible spectrum. (After Chapanis, Garner, and Morgan.[3])

The stimulus can be varied in three ways: in wavelength, in amount, and in homogeneity of the light. Reference to Figure 13.4 will show the variation of wavelength possible between the limits of visibility. It should be emphasized, however, that the wavelengths themselves are not colored; the problem of color is a psychophysical one which we shall discuss shortly. The stimulus may also vary in amount of radiant energy present at each wavelength — in everyday terms, the light may be dim, moderate, or very bright. For precise definition, the amount at each wavelength must be specified, but for some purposes it is sufficient to indicate the total amount of light emitted by a source, or falling on a surface, or reflected from a surface. The third physical variable is homogeneity of the light, that is to say, the distribution of wavelengths present in it. A homogeneous light is one in which only a very small band of wavelengths occurs —

say one or two millimicrons wide. A nonhomogeneous or heterogeneous light is one in which many wavelengths are present, perhaps even the entire visible spectrum in various amount, as in daylight. We shall see what these various conditions mean in terms of vision, but at present we are only interested in the stimulus itself and in the ways in which it can be described. It is necessary, then, that a stimulus be specified in terms of wavelength, amount of radiant energy present in each wavelength, and the relative homogeneity of the light. White light is a rather complex psychophysical problem, but the term is loosely used to mean any light which is physically complex (exceedingly heterogeneous) and produces no obvious or strong color for a normal observer. White light usually contains most of the wavelengths to which the human eye is most sensitive.

Psychological Dimensions and Relationships. If we now turn to the purely psychological dimensions of vision — what we see and the ways in which this can be independently varied — we must forget, for a moment, all about the stimulus. The psychological dimensions are three in number also, but it must not be assumed that these have any necessary relationship to the physical variables — that is something which we must investigate later. A visual sensation, a " **color,**" may vary in **hue**, in **brightness**, and in **saturation**, and any of these may be varied independently of the others. For an accurate description of a color, then, each of these must be identified. Hue is that aspect of color which the layman usually means when he says " color." It is the redness, blueness, greenness, yellowness, and can be defined only by immediate reference to an example. I point to the sky on a clear day and say, " That is what I mean by blue." Brightness is that aspect of color which is represented by the difference between a 25-watt bulb and a 200-watt bulb in a lamp, or between a light blue sweater and a dark blue sweater, or pastels versus colors. Saturation is that dimension which can be used to describe the difference between the color of the ocean on a bright, hazy day and on a clear day — gray-blue versus clear blue of about the same brightness. It is the grayness of a color.

Figure 13.5 shows a schematic representation of the three psychological dimensions and their relationships. All the space inside the color solid represents possible colors. It is apparent that one may go in any one of the three dimensions without changing the other two. One can vary hue by going around the color solid at the same level on the black-white (B-W) dimension and keeping the same distance from the center. One may vary brightness by moving up and down parallel to the B-W dimension, but not moving around the hue circle or varying the distance from the B-W line. Finally, one may vary the saturation by moving in towards the B-W line at the same level, without moving around the hue circle. Thus, the dimensions are conceptually independent, although it may prove extraordinarily difficult to achieve this ideal situation in actual practice. Note, for future reference, the spatial relationships of the seven key points on the diagram. Black is opposite white, red is opposite green, and yellow is opposite blue. The exact center of the diagram is a point called neutral gray — it has no opposite. The color solid also shows the important psychological fact that it is impossible to have highly saturated colors which are at the same time very light or very dark. As one moves toward either the black or the white pole on the outside of the solid, at the same hue, one moves closer and closer to the B-W line, until saturation is entirely lost with colors of extreme lightness or darkness. This is exemplified by the difficulty in distinguishing between " midnight " blue and black.

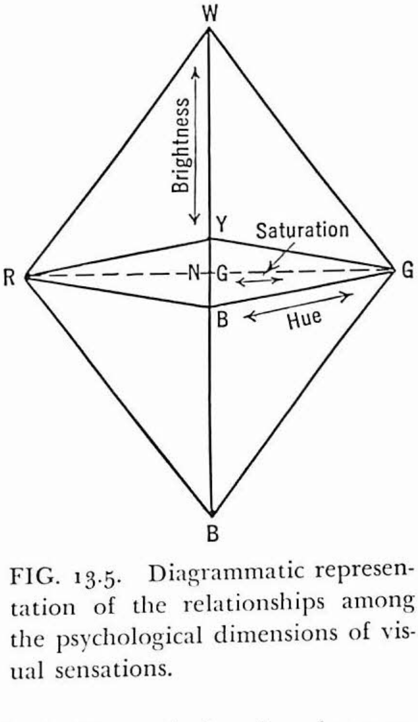


FIG. 13.5. Diagrammatic representation of the relationships among the psychological dimensions of visual sensations.

The seven key points on the diagram are said to be the " psychologically unique " colors, in terms of which visual sensations may be described. The red, for the observer, is one which is neither yellowish nor bluish, the blue is one which is neither reddish nor greenish, and so on. The white must be the brightest possible color, with no hue, and neutral gray must be no blacker than it is white. A psychological description of a patch of color might thus be as follows: a yellowish red of medium brightness and low saturation (it would usually be called brown). The Munsell color system is a standardized reference system, based upon these three dimensions, which provides sample colors at many points. All one needs to do is to match one's own sample with one of the Munsell chips and the approximate description can be read off directly. It takes considerable practice to describe colors even reasonably accurately, so such systems are great time-savers.

The Psychophysics of Vision. We are now in a position to consider the relationships that exist between the stimulus dimensions and the psychological dimensions. These relationships are not simple. In general, one may say that hue is primarily correlated with wavelength, brightness is primarily correlated with amount of radiant energy, and saturation is similarly related to homogeneity of light. However, these relationships are rough at best and many factors enter to modify them.

If we state that, in general, hue is related to wavelength, we must take account of the factors which modify the statement. It will be immediately obvious that for the color-blind or color-weak individual the normal relationships (indicated in Fig. 13.4) do not hold. It is also true, as we shall see, that in after-images there is hue without a stimulus which can be specified in terms of wavelength, and the state of adaptation is an important determiner of hue. In dim light, where the rods alone are functional, there is no hue at all, regardless of the wavelength of the light, for the rods are completely color-blind. Hue can also be modified somewhat merely by changing the amount of radiant energy.

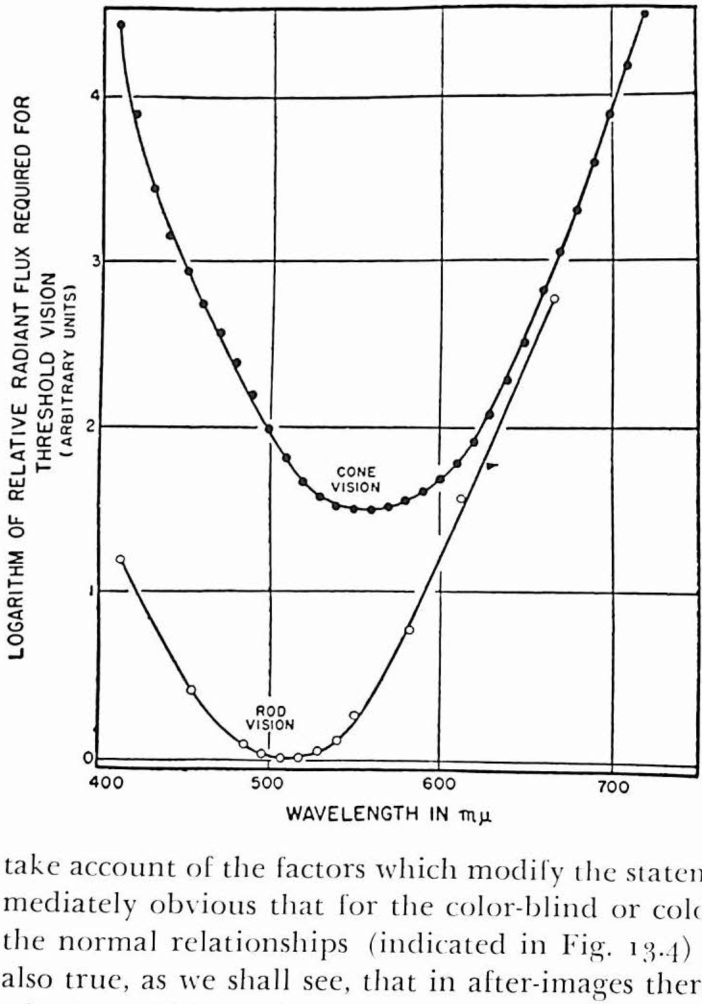


FIG. 13.6. Photopic (cone) and scotopic (rod) visibility curves showing the relative sensitivities of each to light of various wavelengths. (After Chapanis.[5])

The statement that brightness is correlated with amount of radiant energy must be similarly modified, for brightness is easily seen to be related also to wavelength. Wavelengths beyond the visible spectrum demonstrate this nicely. With very short or very long wavelengths, there is no stimulus which will excite the receptors. No matter how great the amount of radiant energy, there is no brightness, for the energy is not visible. Moreover, there is a variation in the brightness produced by the same physical energy at various wavelengths within the visible spectrum. Figure 13.6 shows this function in terms of thresholds. The most effective wavelengths in terms of producing brightness for the normal human eye are those in the neighborhood of 555 millimicrons for the cones, and about 511 millimicrons for the rods.

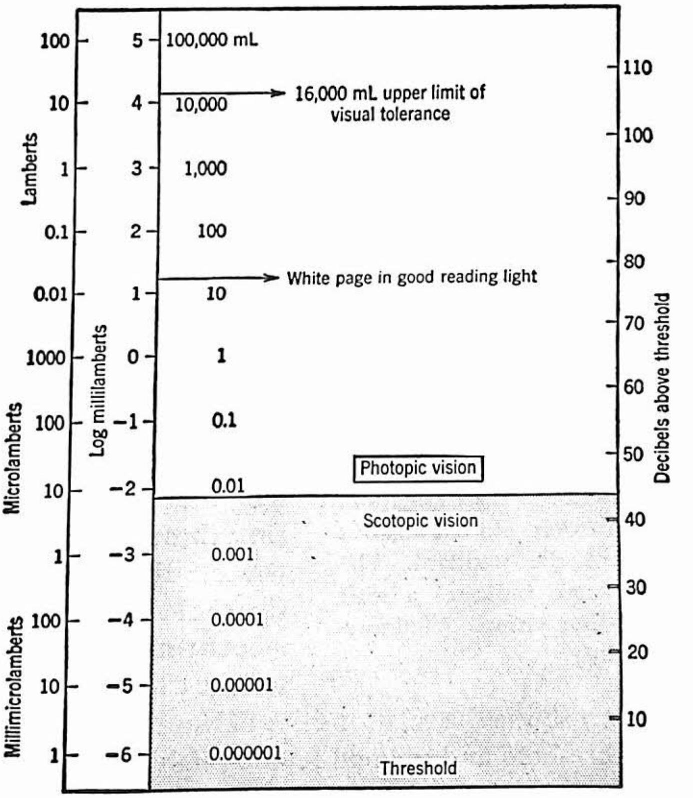


FIG. 13.7. Diagram of human visual thresholds. (After Bartley.[6])

Finally, we also modify the statement that saturation is dependent upon homogeneity of light, for with dim light, where rods alone are functioning, saturation is zero even with the most homogeneous light. Even with the cones operating, saturation is dependent upon amount of radiant energy to the extent that dim or very bright light reduces saturation; the best saturations are obtained with moderate amounts of radiant energy. And some wavelengths produce more saturated hues than others; the yellows are less saturated than the other primary hues.

Visual Thresholds. It has been calculated that the actual threshold for the stimulation of one rod, under optimal conditions, is one quantum of radiant energy, the smallest measurable unit of light.[4] However, due to a number of factors, a larger amount must enter the eye and several rods must be stimulated for sensation to occur, so that experimental results yield thresholds a good deal larger than this. As one would expect from variations in the fineness of the mosaic of the retina and from variations in the number of diffuse connections over a retinal area, the absolute threshold is dependent upon the size of the stimulus and the retinal area stimulated, as well as by the wavelength of the stimulus and the time through which it acts. For white light the minimal threshold for the rods is about .000001 **millilambert,** whereas for the cones it is .01 millilambert. Figure 13.7 shows the relationships of the absolute thresholds to the terminal threshold and to good reading light.[6]

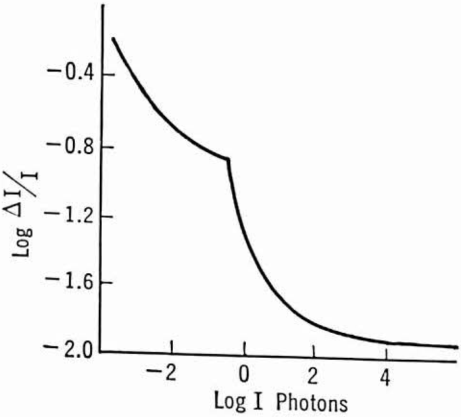


FIG. 13.8. Brightness discrimination. The Weber fraction plotted against the intensity of the standard. The break in the curve indicates a shift from rod to cone vision. (Redrawn from Steinhardt.[7])

For homogeneous light, the threshold varies greatly with wavelength, as we have already noted, and is not the same for rods and cones, although the shape of the curve is similar (Fig. 13.6). The curve for **photopic** vision (cone vision) lies above the other and reaches its lowest point at 555 millimicrons (yellow-green). Light of this wavelength appears brightest to the light-adapted human eye. The curve for **scotopic** vision (rod vision) shows the lower thresholds which we have already come to expect of rods and reaches its lowest point at 511 millimicrons. This represents a shift toward the " blue " or short-wave end of the spectrum (511 is nearly pure green for the cones) and corresponds to the fact that the brightest colors for rod vision are the blues and blue-greens (although, of course, they are not seen as colors, but as light grays). It should also be noted that the long-wave end of the spectrum disappears rapidly with decreasing illumination; reds become black very quickly as the light fades. At twilight, reds and oranges darken as greens and blues become relatively lighter. This effect, called the **Purkinje phenomenon,** represents the shift from cone to rod vision and can easily be predicted from the curves in Figure 13.6.

In vision, three kinds of difference limen are important: those for brightness, those for hue, and those for saturation. As in the case of absolute thresholds, many factors can be expected to influence the brightness limens. Figure 13.8 shows a typical curve for brightness discrimination. There is an increase in the Weber fraction as the intensity increases; it is not a straight line function. The curve is discontinuous because of the shift from rod to cone vision. Discrimination can be seen to be very fine for low intensities and relatively gross for high intensities. There are over 500 JND steps in the brightness discrimination scale.

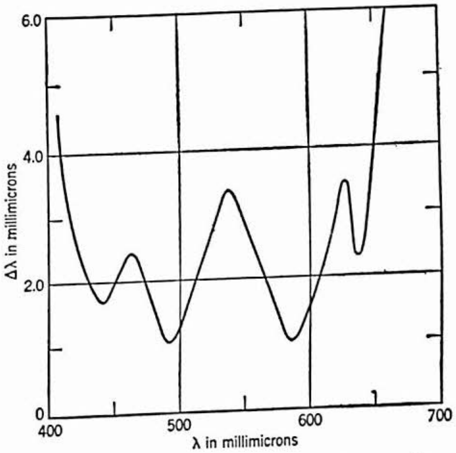


FIG. 13.9. The variation of hue discrimination with wavelength. (After Geldard.[8])

The discrimination of hue is not a constant function but varies with wavelength, as Figure 13.9 shows. The finest discriminations occur in the blue-green and orange regions, where it is of the order of 1 millimicron, the poorest in the extreme spectral reds and violets. These data do not show discrimination in the purples, since they are not found in the spectrum but are formed by mixing red and blue lights. There are about 128 JND steps in the spectral hue series and about 28 in the purples. Depending upon the hue, there are somewhere between 16 and 23 JND steps in the saturation series for a given hue. A rough approximation of the number of discriminably different colors, taking all dimensions into account, has been estimated as about 10 million.

Perimetry and the Color Zones of the Retina. It is obvious from the structure of the retina itself that visual acuity will be less as one goes from the fovea toward the periphery. It is not obvious that there are also changes in color responses which go hand in hand with these changes. **Perimetry** refers to the investigation of areal differences in the retina by means of an apparatus which permits small test objects to be moved along known courses. If test objects of various colors are used, it is found that the extreme periphery is color-blind and that the blue and yellow fields are more extensive than the fields for red and green. Figure 13.10 shows the extent of each of the fields for standard conditions. The exact extent of the fields depends upon a large number of factors, among the most important of which is the intensity of the stimuli. These results do not hold for extremely small or extremely large test objects. Standard test objects will be seen to change color as they are brought in from the extreme periphery. An orange disc will first appear as gray, then will become a yellow, and finally will appear orange, as it enters the smaller red field. Similarly, a blue-green disc will first appear gray, then become blue, and finally blue-green. One is not aware of this change in color in the normal course of events, any more than one is aware of the blind spot; we usually look directly at an object, and an object which overlaps several zones is seen as a unit and in its "best," or most central, color. There are only four invariable colors, or physiologically

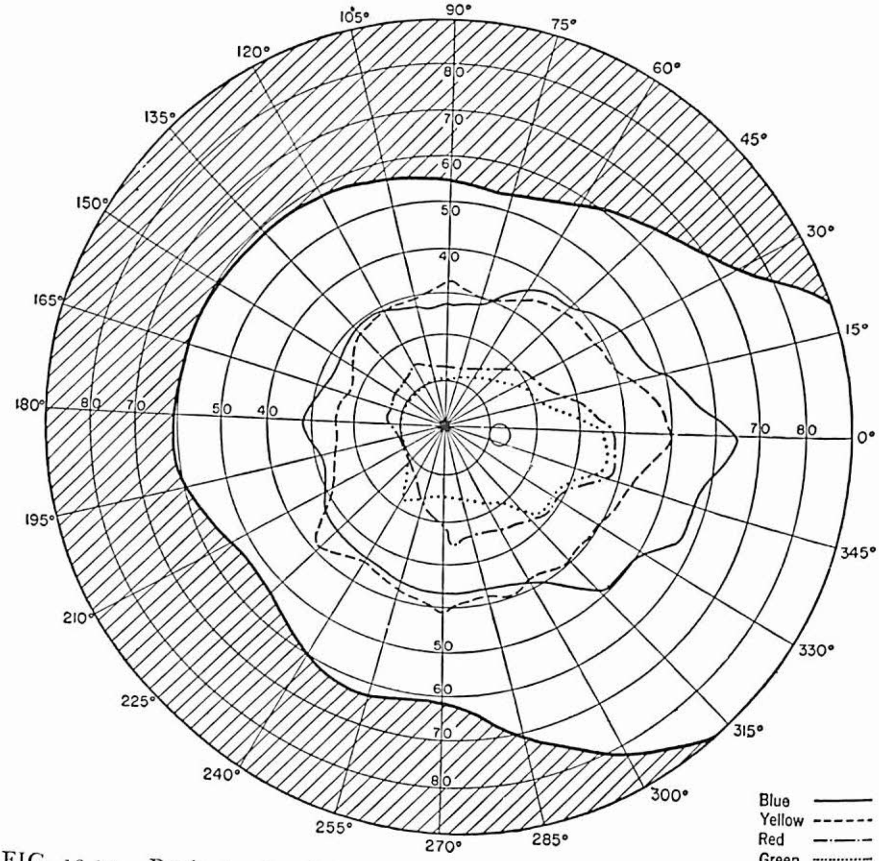


FIG. 13.10. Retinal color fields of right eye with a small test object of moderate intensity. (After Dimmick.[9])

unique colors, as they are often called: a red, a yellow, a green, and a blue. These do not change in hue but are first seen as gray, and then change to their proper direct-vision color without going through an intermediate step. These "physiological primaries" do not correspond exactly with the "psychological primaries," unfortunately for color theory.

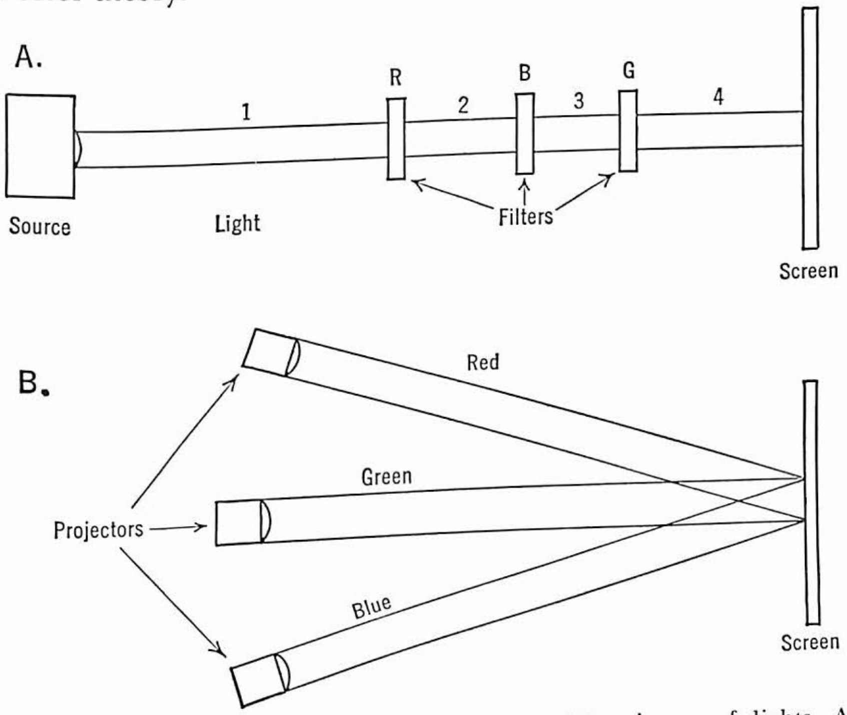


FIG. 13.11. The subtractive (A) and additive (B) mixture of lights. A. Light from the source (1) is white. Red filter passes longer wavelengths only, so light at (2) appears reddish. Blue filter passes only the shorter wavelengths, but since the light (2) falling on it contains only long wavelengths, it can pass only a few middle wavelengths, so the light at (3) contains only these and appears yellowish green. Green filter passes the middle wavelengths, and the light at (4), falling on the screen, is thus yellowish green. B. Light from each source is added to the total. The result here is white.

Stimulus Mixture. Stimulus mixture in vision is a rather complicated topic which is of great importance industrially, because of the problems of matching colors, as in paints and yarns, and of the use of color standards of quality in the grading of some products, such as tomatoes and paper. There are basically three kinds of mixtures: additive mixtures of lights, subtractive mixtures of lights, and mixtures of pigments. Mixture of pigments is an empirical affair, the results depending upon the particular pigments in the paint, and has no generality of application. Subtractive mixture of lights occurs when light passes through successive layers or filters, each one passing only a selected band of the spectrum. The light falling on each successive layer contains only those wavelengths which were not subtracted by the preceding layer. Figure 13.11A shows the principle involved in subtractive mixture. It is not of great importance but should be understood, since interposing two filters in a single beam of light will give results of this sort. It is the basis of the color mixture which occurs in color film where three layers, each a different color, are used to " match " all the colors of a scene.

The additive mixture of lights is shown in Figure 13.11B. In this procedure, each beam of colored light is independently superimposed upon the same spot, so that all the wavelengths in every beam are still present; in other words, they are added together. Additive mixture also occurs in newspaper color pictures composed of fine dots of three colors and in color television, where fine lines of three different colors are not resolved by the eye because they are below the limit of visual acuity and so are perceived additively as a single unit. Mixture produced by the rapid rotation of a disc with sectors of variously colored papers, as is the usual method in demonstrations in the classroom, produces almost the same results as additive mixture of lights and the same principles apply for hue and saturation. Speaking on a strictly psychological level and referring to Figure 13.5, one may express the results of additive mixture very generally as follows. Stimuli which give adjacent hues on the color square of Figure 13.5 will, when mixed, give hues which are intermediate between them; thus, red and yellow give orange, green and yellow give yellow-green, and so on. Stimuli which give nonadjacent hues on the color square do not, when mixed, result in intermediate hues, but merely reduce saturation; thus, certain pairs of blue and yellow give gray, and certain pairs of red and green also give gray. The red and green pair and the blue and yellow pair are said to be **complementary**, that is, when the stimuli are mixed, they give **achromatic color:** gray or white. Such colors are opposite each other on the color square; every color on the color square has a color opposite it which is its complement. For every color there is some other color which will mix with it to give gray. This statement holds true for the entire color solid, since mixture can also compensate for variations in saturation and brightness. In the same sense, black and white are complements, because when mixed they give gray. The saturation of mixtures is always lowered, for the light is less homogeneous (see stimulus correlate of saturation). The rule for brightness is different for lights and for colored papers. For lights, brightness must always increase with mixture because one is actually adding more energy to the system (see stimulus correlate of brightness). For papers, brightness averages; alternating two sectors of different brightness contributes less total energy to the system than does a disc entirely covered with the paper of the higher brightness.

The results obtained by the additive mixture of lights have been important in two respects — in the practical problems concerned with the specification of color matches and reproduction of color, and in the theoretical problems concerned with theories of color vision. It is presumably possible to match all hues by the additive mixture of three properly chosen, homogeneous, spectral lights. Figure 13.12 shows the amount of each of three standard primaries required to match all spectral hues for normal observers. It is assumed that, because of this, only three kinds of cones are necessary to produce normal color vision. It should be noted that the curves in Figure 13.12 dip below zero in some places, indicating that a negative amount of that primary is required for matching spectral hues in that region. In terms of stimulus mixture, this merely means adding a small amount to the light to be matched. It is not clear what it could mean in terms of real physiological processes. Furthermore, the matching of all dimensions of color is not perfect; in particular, it is impossible to match saturations to any great extent. This topic will again be considered in the section on theories of color vision.

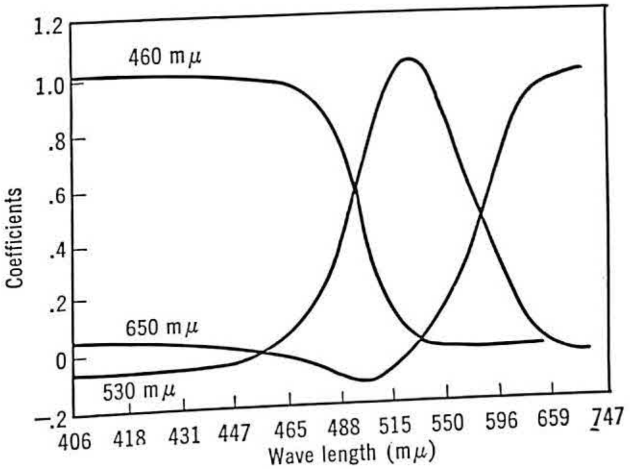


FIG. 13.12. The relative amounts of each of three primaries (650, 530, and 460 mμ) needed to match all spectral colors. (After Wright.[10])

Suggested Readings

1. Geldard, F. A., *The Human Senses*. New York: Wiley, 1953, Chaps. 2, 3, 4, and 5, pp. 14–93.
2. Chapanis, A., Garner, W. R., and Morgan, C. T., *Applied Experimental Psychology*. New York: Wiley, 1949, Chap. 4, pp. 67–117.

14 | Vision: continued

Adaptation. In vision there are two aspects of adaptation which are of importance: brightness adaptation and chromatic (color) adaptation. The first of these is the more obvious. It consists of two related processes, dark adaptation and light adaptation; in other words, it is concerned with level of illumination. Both processes are illustrated when one attends a movie in the daytime. Upon entering the darkened theater, one can see nothing because he is adapted to daylight, a high level of illumination, and is said to be light-adapted. After a period of time, seats and people become visible and more and more detail develops. This is the process of dark adaptation and at the end of a period of time in dim light, one is said to be dark-adapted.

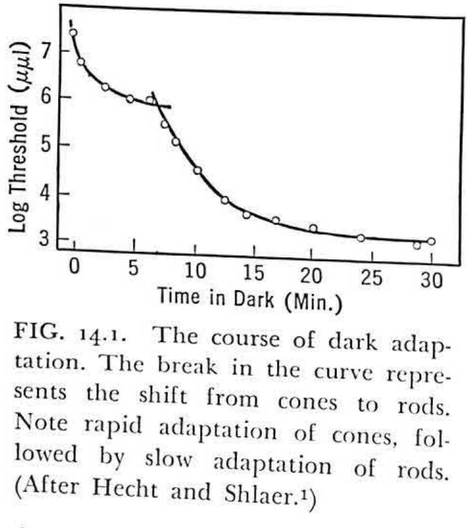


FIG. 14.1. The course of dark adaptation. The break in the curve represents the shift from cones to rods. Note rapid adaptation of cones, followed by slow adaptation of rods. (After Hecht and Shlaer.[1])

Figure 14.1 shows the course of dark adaptation, following adaptation to a high level of illumination and using a test light of short wavelength. The major features of normal dark adaptation may easily be seen. There is a gradual increase in sensitivity (the thresholds become lower) as time in the dark increases. Dark adaptation is almost complete in about 30 minutes, although slight changes can be shown even after this. The break in the curve at about 7 minutes represents the end of the adaptation of the cones — they adapt rather quickly but do not function at all in extremely dim light.

Figure 13.7 showed the level at which vision becomes the exclusive affair of the rods (scotopic vision). The visual pigment in the rods, rhodopsin, is bleached by light and is built up in the dark. The long course of dark adaptation is related to the time necessary to rebuild this visual pigment. The greater the intensity of the previously experienced illumination and the greater its duration, the longer it takes to build up a sufficient supply of rhodopsin. A day at the beach, with its tremendously high level of illumination, will interfere significantly with the level of dark adaptation which one is able to reach that evening. On the other hand, if one is fairly well dark-adapted, driving along a lonely road, the light from an on-coming car produces only a brief loss in sensitivity, because the time during which the lights are seen is brief.

As Figure 13.6 showed, the rods do not have the same visibility curve as the cones; it is displaced toward the short-wave end of the spectrum. The rods are relatively more sensitive to light of short wavelengths and much less sensitive to light of long wavelengths. To the dark-adapted eye, reds appear black (i.e., red does not stimulate) and blue-greens and blues appear light gray. A curve like that in Figure 14.1, but drawn for light of long instead of short wavelength, would show only the first part of the curve, that for the adaptation of the cones. Similarly, a curve for a stimulus which is small and confined to the fovea would show only the first part of the curve, for there are only cones in the central fovea and very dim objects cannot be seen there.

There are three parts to the process of dark adaptation. First, the pupil enlarges rapidly, admitting more light to the retina. Secondly, the cones become more sensitive during the first 7 to 10 minutes in the dark. Finally, the rods become very much more sensitive during the first 30 minutes or more in the dark. The total increase in sensitivity is about 10,000 times.

Light adaptation is the reverse process. The pupil contracts, reducing the amount of light admitted; the cones become less sensitive; and the rhodopsin in the rods is bleached, very much reducing their sensitivity. Above .01 millilambert the rods do not function and we have photopic vision. Light adaptation takes place relatively rapidly; a very few minutes are required to reach substantially complete adaptation.

Vitamin A is important in dark adaptation because it is the raw material from which rhodopsin is made. Vitamin A deficiency has been shown to be related to inability of the rods to recover after exposure to light. An individual with extreme vitamin A deficiency shows only the cone adaptation curve and is said to be " night-blind." Night blindness is obviously important in driving, or even walking, at night.

There are many situations in which the greatest possible sensitivity in dim light is vital. This is the case in night flying, particularly combat flying, where ability to see a little better than the enemy makes all the difference. Flyers scheduled for night combat must be dark-adapted before they take off. Since sitting in a totally dark room for a half hour can be very boring, as well as a waste of time, red goggles are often used. Reference to Figure 13.6 will show that the rods are very insensitive to " red " light, whereas the cones are much more sensitive. Red goggles permit one to see, by cone vision, well enough to go about one's duties, but they reduce the amount of light which is stimulating to the rods almost to insignificance. For the same reason, using red glass on lighted dials in the cockpit will interfere less with the course of dark adaptation than will glass of any other color. This fact is also made use of in the navigation of vessels at night, where the utmost in sensitivity is essential to see hazards of many sorts. Some charts are so printed that they may be read under red light, thus enabling the navigator to check his position without making him temporarily night-blind.

Chromatic adaptation deals with qualitative changes, rather than with general level of sensitivity. It refers to the fact that, other things being equal, colors tend to become grayer or more " washed out " with continuing stimulation. If, on a gray day when the daylight is not strongly colored, one walks into a room which is illuminated by ordinary incandescent bulbs, one is impressed by the definite yellowness of the light. After a period of time in the room, however, the yellowness disappears. Chromatic adaptation has occurred; the yellow has become desaturated, has become grayer, has lost its hue. The same phenomenon occurs with a small spot of color. If you stare at it without moving your eyes, it gradually becomes less and less saturated, it appears washed over with gray. This effect is more pronounced and more complete with colored general illumination and with colors which are not highly saturated to begin with, partly because it takes a long time to adapt chromatically to saturated colors and, also, because one cannot keep his eyes perfectly still. But recently complete chromatic adaptation to a rather saturated stimulus has been shown to occur for a homogeneous visual field. " White " light, which is hueless, is the only illumination which does not have this effect and is, therefore, essential for color matching or color judgments. Many modern stores attempt to provide neutral illumination which avoids to a large extent chromatic adaptation, and also distorts the colors of objects less.

After-sensations. Whereas adaptation refers to the effect of stimulation while it is occurring, after-sensations refer to the effect produced after the stimulus is removed. The term " after-image " is frequently used interchangeably with " after-sensation." In either case, the reference is to the persistence of sensation after the removal of the stimulus. After-sensations occur in many senses, but nowhere are they as many and as varied as in vision. There are three distinct types of visual after-sensations, usually corresponding to different kinds of original stimulation, though several may occur in response to one stimulus. With very intense light, "**flight of colors**" occurs — the stimulus is followed by a changing sequence of purples, blues, yellows, greens, reds, and so on. The exact sequence and duration vary. Flight of colors follows looking directly at the sun, for example, though this stimulus is so intense that if the duration is anything but very short, the after-sensations may persist for a long time. With moderately intense but very brief stimulation, **positive after-sensations** may be observed. As the name implies, a positive after-sensation is one which is just like the original sensation; it has the same hue and brightness relations. It is usually fleeting and hard to observe without practice. It can best be seen by looking at a brightly lit sky from a fairly dim room through a window that has some bars or cross pieces. Then, if one opens his eyes and closes them again rapidly, he will see a very brief flash of bright sky and dark bars just after he closes his eyes. Often the positive after-sensation is followed by the **negative after-sensation,** in which the hue and brightness relations are reversed.

Typically, the negative after-sensation follows a long stimulation with moderate or weak stimuli. Every color appears as its complement; red areas in the original stimulus become green in the after-sensation, whites become black, only neutral gray does not change. In the above example of a positive after-sensation, one would ordinarily find it followed by a negative after-sensation in which the sky appears as a black patch and the bars as light. A more usual situation for the observation of negative after-sensations is staring steadily, without moving the eyes, at a small patch of color for about 30 seconds, and then staring at a piece of gray paper (a neutral wall will do for a gross demonstration). After a few seconds, the negative after-sensation "develops," but it will disappear temporarily if the eyes are moved. It is obvious that such after-sensations can interfere with color matches and it is for this reason that in experimentation with vision a neutral gray background is essential. Although we do not normally notice even these persistent after-sensations, because we seldom have our eyes still, they nevertheless influence judgments of color. A totally acceptable explanation of after-sensations has not yet been forthcoming. They seem to be related to the process of recovery from stimulation, presumably chemical in nature because of the relatively long recovery period.

The existence of after-sensations is one of the strong arguments for not equating wavelength with seen color. What wavelength corresponds to the after-image of a patch of homogeneous light of wavelength 505 mμ? For that particular red image at that particular moment there is no externally measurable stimulus, yet the color is most insistently there. Again, the most saturated colors ever seen are those in which an after-sensation is projected upon a background that is the complement of the original stimulus; e.g., the after-image of a red patch projected upon a piece of green paper, the exact complement of that red. The colors obtained in this way are far more saturated than any homogeneous spectral light, although they cannot be directly related to an external stimulus.

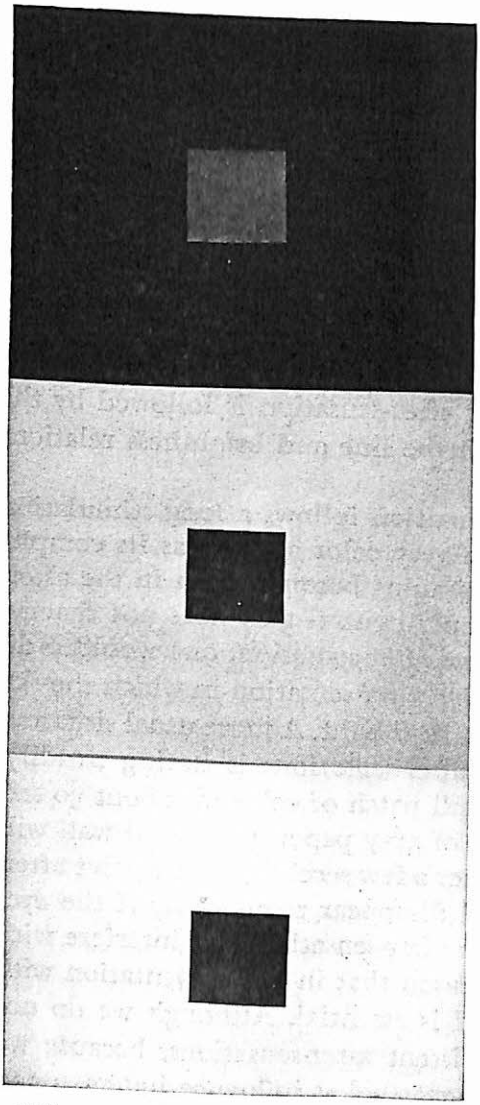


FIG. 14.2. Simultaneous contrast. The three small squares are the same brightness and were cut from a single sheet of gray paper. They appear different because of the surround.

Simultaneous Contrast. Simultaneous contrast, often just called contrast, refers to an ever-present effect in any patterned visual field — each area of color affects the region around it. The effect is such that it tends to increase the visibility of the pattern. Sometimes the effect is summed up in a kind of law. Every color tends to induce its complement in its surround. Simultaneous contrast is thus related to negative after-sensations (which are sometimes called successive contrast) because it shows the same phenomena in a spatial pattern that a negative after-sensation shows in a temporal pattern. Reference to Figure 13.5 will give the basic pairs of complements from which one can predict what any induced color (the color induced in the surround by the test patch) will look like. Note that black and white are also a part of this phenomenon. For black, this contrast effect is essential, for the seeing of black depends entirely upon it. Black is a contrast color; that is, one never sees black unless it is induced by a white (or very light) surround. The complete absence of light, as would obtain in a light-proof room with the eyes dark-adapted (to permit dissipation of successive contrast or after-sensations of light), does not give black psychologically; it gives a rather dark gray. The startling, crisp, velvet black which is the standard black is obtained only by surrounding a tube lined with black velvet by a highly reflecting paper which has very high illumination falling on it. The intensity of the black is directly related to the whiteness of the white, i.e., it is induced by the white, and is its complement.

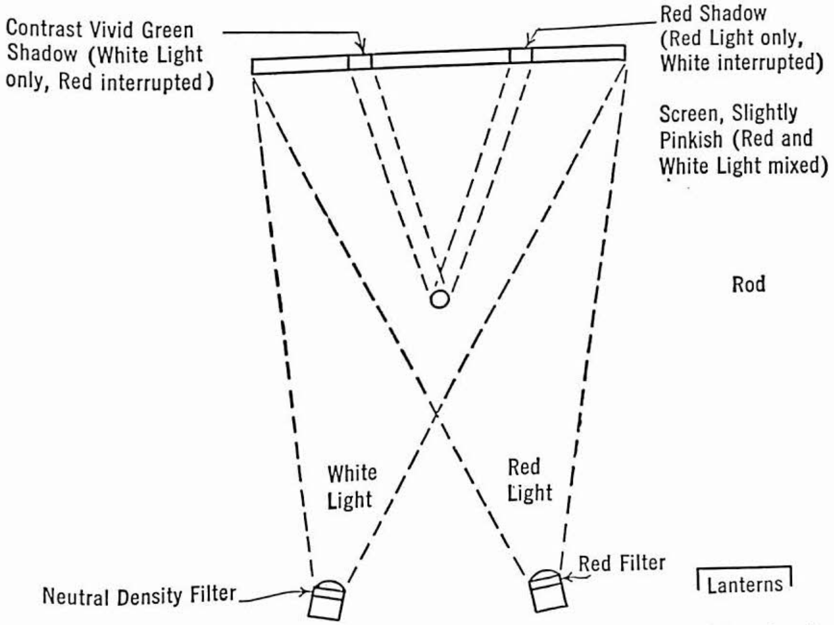

Contrast Vivid Green Shadow (White Light only, Red interrupted)
Red Shadow (Red Light only, White interrupted)
Screen, Slightly Pinkish (Red and White Light mixed)
Rod
White Light
Red Light
Red Filter
Lanterns
Neutral Density Filter

FIG. 14.3. Situation for the production of " colored shadows " by simultaneous contrast (called the Hering window).

Figure 14.2 shows this effect, though not as strongly as it can be shown with the great differences in illumination possible in the laboratory. " Colored shadows " are the result of contrast and under experimental conditions the induced color is unbelievably saturated. It is possible that there is an inhibitory process in the retina which influences saturation. Figure 14.3 shows the situation necessary for the production of colored shadows. It sometimes occurs outside the laboratory — for example, utilizing reflected green light from grass as one source and sky light as the other, with the production of light pink shadows.

Whether one is aware of the contrast effect or not, it enters into the perception of colors; a blue is made more saturated by the proximity of a yellow patch, a complexion is made more ruddy by wearing a green sweater. Since both chromatic and achromatic colors enter into contrast effects, these are often discussed separately under chromatic contrast and brightness contrast, although the principles can be understood by merely considering black and white as complements. However, brightness contrast has a place of special importance in the topic of visibility. We shall refer to it in greater detail when we discuss visual acuity. It has been found that for legibility of signs, for example, maximum brightness contrast is desirable; one would not use light green lettering on a soft gray ground for highway signs. But a certain amount of chromatic contrast also helps, if it does not reduce brightness contrast too much. The best visibility in signs thus turns out to be yellow on black, combining high brightness contrast with some chromatic contrast.

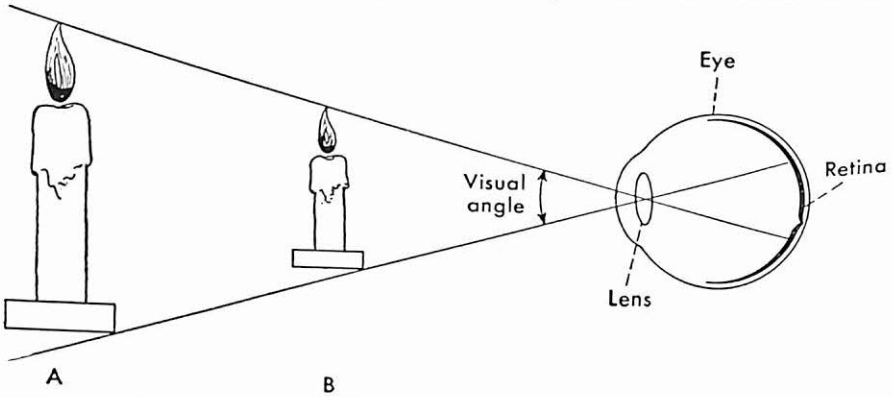


FIG. 14.4. Diagram showing visual angle. Visual angle is the angle between the two lines drawn from the center of the lens to the top and bottom of an object. It is measured as any angle is measured, in degrees, minutes, and seconds. Note that the larger object, A, which is further away, subtends the same visual angle as object B – size and distance are interchangeable.

Visual Acuity. **Visual acuity** is an important aspect of the perception of pattern in visual space, for it sets limits to the fineness of perceptible pattern. It may be defined as the smallest spatial pattern which is discriminable. Usually, it is defined in terms of the **visual angle** of the smallest visible object, which takes account of both the size of the object and its distance. It is the angle made by two straight lines drawn from the lens of the eye to the top and to the bottom of an object (cf. Fig. 14.4). There are very many factors which influence this threshold, and the psychophysiological theory of visual acuity throws some light on perception in general and the manner in which the nervous system must work in visual discrimination.

There are a number of ways of measuring visual acuity and they do not give exactly the same results because of the large number of factors which influence the threshold. The most familiar measure is the **Snellen chart** used by doctors in testing the eyes for glasses. Letters of various sizes are used at a standard distance and the visual angle subtended by each varies as it would if letters of identical sizes were moved closer or farther away. If one can just see at 20 feet what the average person with good vision can see at 20 feet, then he has 20/20 vision. Many people have better vision than this, being able to see at 20 feet what others see at 15 feet (20/15 vision); and many have poorer vision, being able to see at 20 feet what others can see at 40 feet (20/40 vision). The visual angle subtended by the width of stroke of the letters at the 20/20 point is one minute of arc, i.e., visual acuity is 1.0 at this point. The Snellen chart is a good rough index of visual acuity for distant objects, but it is not adequate for experimental work. It is also interesting that visual acuity is not the same for near objects as it is for distant objects. This is important in some industrial jobs where there is continuous close work. For such jobs, testing of prospective employees for visual acuity must be done at close distances, not with the Snellen chart or other tests of distant acuity.

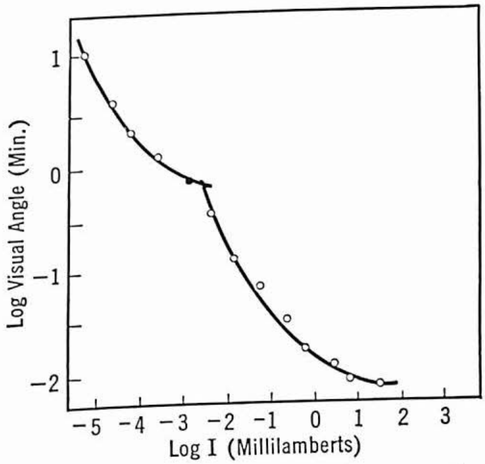


FIG. 14.5. The relationship between the brightness of the background and the visual angle subtended by a just visible wire. As the brightness of the background is increased, the threshold visual angle decreases, or the visual acuity increases. (After Hecht and Mintz.[3])

Among the many test objects which have been used in measuring visual acuity for scientific purposes are Landolt's broken ring (or C) and a very fine wire. With both of these, thresholds in terms of distance from the eye may be obtained under a variety of conditions. With the Landolt C, one obtains a threshold for the minimum separable, or the visual angle subtended by the break in the circle. Ordinarily this turns out to be about one minute of arc, but under special conditions and with very high brightness, a figure as low as 24 seconds has been obtained.[2] The fine wire technique, in which a single fine wire is seen against a bright ground, yields much lower thresholds; one-half second is not uncommonly found.[3] The threshold here is related to the length of the wire; for very short wires (a dot, as the extreme example) the thresholds are much higher, of the order of 30 seconds,[2] thus showing the importance of spatial summation in acuity.

Among the factors which influence visual acuity, one of the most obvious is the level of illumination. Visual acuity improves rapidly as the level of the illumination is increased. Figure 14.5 shows the decrease in visual angle subtended by a fine wire at threshold as the illumination increases. The break in the curve represents the shift from rod to cone vision. In making measurements of this sort, the subject must be adapted to the level of illumination before each test. Quality of illumination is also important. White light and monochromatic light from the middle of the visible spectrum give the best acuity; light from the extremes of the spectrum results in lower acuity.

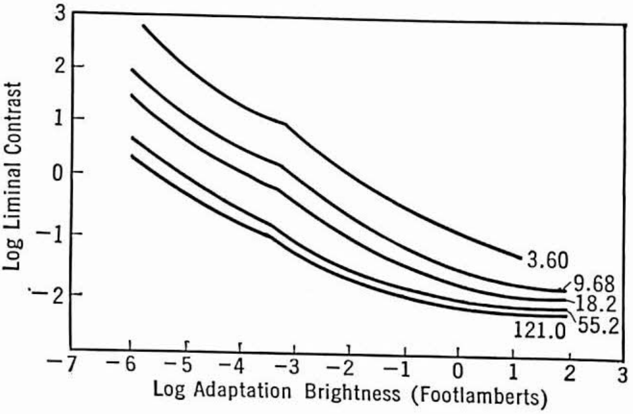


FIG. 14.6. Threshold showing the amount of contrast necessary (log liminal contrast) to be just perceptible at different levels of field brightness (log adaptation brightness) for five stimulus areas. (After Blackwell.[4])

Contrast is as important for the measurement of visual acuity as it is in the design of highway signs. An extensive study of the relation of field brightness to contrast has shown a regular decline in the amount of contrast necessary for visibility as the field brightness increases. Conversely, an increase in contrast is necessary as the brightness is decreased. Figure 14.6 shows these results. It makes very little difference, in most cases, whether the stimulus is dark on a light ground or light on a dark ground.

The importance of contrast in visibility is put to good use in the art of camouflage. Here objects are made to blend with their surroundings by reducing contrast to a minimum (both brightness and chromatic contrast in this case), and the contours which give objects their recognizable shapes are destroyed by putting in "false" contrast in the wrong places and eliminating it where it normally occurs. Another place in the modern world where these facts are of significance is in observation of radar screens. It has been shown that the lowest contrast thresholds and the shortest detection times occur when the illumination of the test field and of the surrounding, or adapting, illumination are nearly equal.[5] Under these circumstances the signal (the information-giving pip on the screen) can be seen most readily. The same principle operates in viewing television, where one sees best with moderate room illumination, not in the dark.

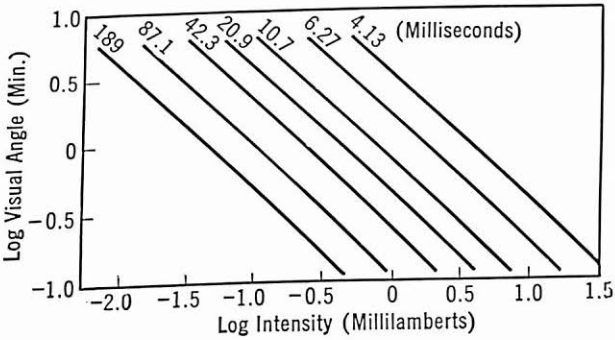


FIG. 14.7. The relation of threshold visual angle to intensity of slit for each of seven durations. As the intensity of the slit increases, the threshold visual angle decreases (the visual acuity increases). The longer the exposure time, the less the intensity necessary for a given threshold visual angle. (After Niven and Brown.[6])

Duration of stimulation is another variable in visual acuity. Figure 14.7 shows how the threshold visual angle at any given intensity increases as the duration of the stimulus decreases. The shorter the time of stimulation, the larger the stimulus must be to be visible, if intensity is held constant.

Size, as a factor in visual acuity, is well shown in Figure 14.6. At every adaptation brightness, the smaller test objects require greater contrast in order to be just visible. Further, the necessary contrast increases more rapidly for the small test objects than for the large as the adaptation brightness decreases — the difference between the large and the small test objects becomes greater as the brightness decreases.

Finally, acuity depends upon the region of the retina upon which the image falls. For photopic vision, maximum acuity occurs when the test object is fixated, when the image falls upon the fovea, for there the cones are slender and tightly packed. Acuity falls off rapidly toward the periphery, as one could predict from the structure of the retina. For scotopic vision, acuity will be zero at the fovea because there are no rods. Scotopic acuity has been found to be greatest at about 4° from the point of fixation and to decrease rapidly as the image moves peripherally. The reason for this is that the rods are gathered into larger groups as one goes peripherally, more rods connect with a single ganglion cell (and intermediate bipolar cell), thus decreasing the discriminative power of the retina. Figure 14.8 shows these effects clearly.[7]

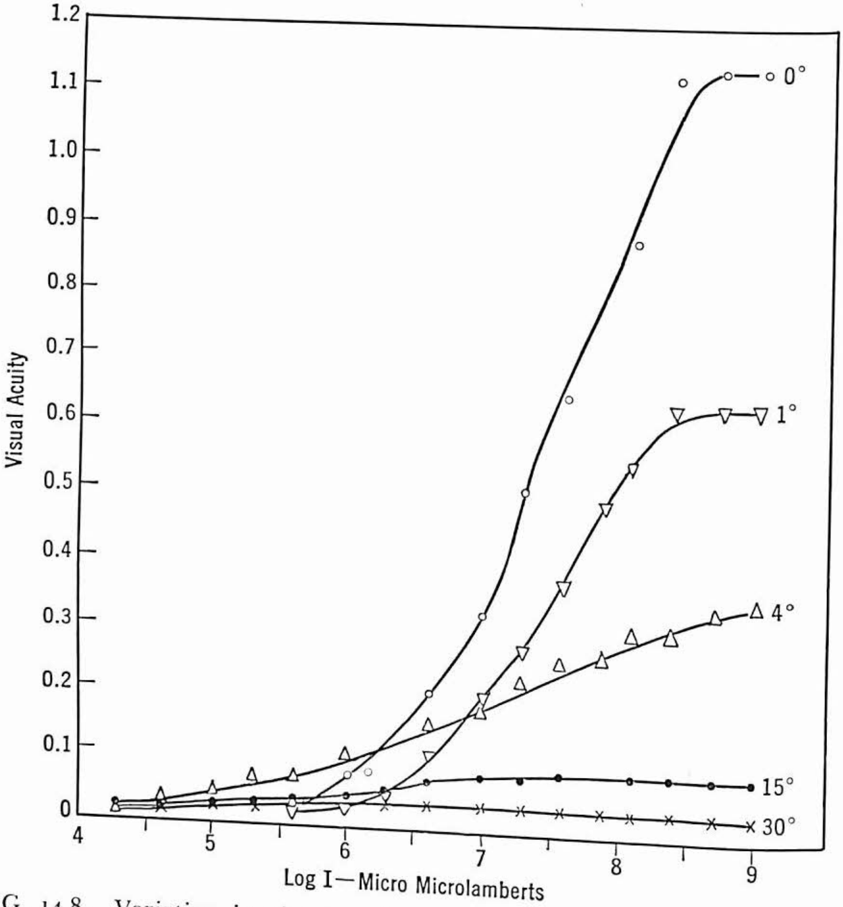


FIG. 14.8. Variation in visual acuity with illumination for five regions of the retina. At 0 degrees (the fovea), acuity is best at the higher illuminations, but at lower illuminations, acuity is best a 4 degrees from the fovea. However, the greatest over-all acuity occurs with high illumination and central fixation. (After Mandelbaum and Rowland.[7])

There are a number of physiological factors which enter into the determination of the limits of acuity in the human eye. In the first place, diffraction or scattering of light occurs at the edges of the pupil. This affects acuity most when the pupil is of small diameter, which is in intense light. Scattering of light also occurs to a significant degree in the material inside the eyeball and this reduces the sharpness of the image because it blurs the contours and reduces the difference in intensity between the image and the surround. This intensity difference is probably the basis for the discrimination of object-no object, upon which the threshold for visual acuity depends. Furthermore, **aberration** is important in the lens of the eye, just as it is in a camera or telescope lens. One form of aberration, called astigmatism, for example, must be corrected by glasses if an individual is to see clearly. Also of some importance is **physiological nystagmus,** involuntary tremors and drifts and jerks which the eyes make even when there is an attempt to maintain perfect fixation. The total extent of movements over a time period of 3 to 4 seconds is usually somewhat less than 10 minutes of arc, but may be much higher. However, perception can occur much more rapidly than this, so that smaller excursions, the 30 to 70 per second oscillations which cover less than a minute of arc, are more influential in setting the limits of acuity. It has been calculated that the image can remain on a single receptor but a few hundredths of a second.[8] Another factor is the " grain " of the retinal mosaic — the size of the anatomical and physiological elements which underlie discrimination. Just as the limitation upon the size of clear enlargement possible from a photographic film is dependent upon the grain of the film, so the grain of the retina determines visual acuity. The finest grain occurs in the central part of the fovea where the cones are most slender and most thickly packed, and also where the physiological unit is a single cone, its bipolar cell, and its ganglion cell, with no sharing of the latter.

These factors all have a place in the over-all consideration of visual acuity, but they do not explain it fully. Foveal cones, in the central part, are 50 times as wide as the image cast by the finest discriminable object. The extremes of visual acuity thus, obviously, cannot be explained entirely by the fineness of the retina, although it is one of the most significant variables. Acuity depends upon slight differences in intensity within the very diffuse image which even a fine line throws upon the retina. It is possible that the sharpening of acuity below the limit of the diameter of a single cone is dependent upon the rapid oscillatory movements of physiological nystagmus. Because of these, temporal summation of brightnesses is possible and, since the cones are not arranged in regular rows, the cones will receive a varying amount of illumination over an interval of time. Also, spatial summation can occur both in the retina itself and in cortical projection fibers. We have seen that a long line is more easily seen than a very short one. This is partly due to the greater opportunity for spatial summation within the retina because of the convergence of several cones upon a single ganglion cell. It may also be accounted for in part by a more complicated system of amplification and suppression at higher levels in the visual system, made possible by the increase in number of fibers at the level of the lateral geniculate bodies in the thalamus and by the increase in the interrelationships possible. Such an arrangement would operate to smooth the boundaries of excitation and to produce a sharper contour. This, together with the temporal summation permitted by virtue of the physiological nystagmus, permits us to see a sharp line where the retinal image is a diffuse band. The sharp contour is generated by the central perceptual processes from peripheral sense data, and the angle subtended by the object can be smaller than the diameter of a cone because of integration over time and space.

Temporal Patterning. Temporal sequences in vision have been investigated extensively and throw some light upon the basic mechanisms of vision. Among the most thoroughly studied of these is **flicker** and related phenomena. Flicker occurs when there is a relatively slow alternation of stimulation, usually light versus dark. This may be obtained by viewing an interrupted white light through a ground glass screen. If such a light is interrupted at gradually increasing speed, one sees the alternation of light and dark become more rapid until a single flickering light is seen and, finally, fusion is attained where a single homogeneous light appears. The point at which fusion occurs is called the **critical flicker frequency** (sometimes called the critical fusion frequency), and is often abbreviated **c.f.f.** The critical flicker frequency is extremely variable and is sensitive to a number of conditions, both within and without the organism. It is sometimes used as an index of physiological changes, as those resulting from oxygen deprivation in high altitude studies, or from loss of sleep. The method can be used with animals and is important in studying the time characteristic of the visual system and, by extension, of synapses in general.

Critical flicker frequency varies with intensity of the stimulation, as reference to Figure 14.9 will show. The increase is almost linear except for the break in the curve representing the shift from rod to cone vision. Fusion occurs at only 4 flashes per second at the extreme low end of the intensity scale and rises to almost 60 flashes per second at the highest intensities. This is in line with the finding that visual acuity (spatial patterning) increases with increasing intensities; temporal discrimination increases under the same conditions. The region of the retina which is stimulated has an influence upon the c.f.f. also. It is higher in the fovea than in the periphery, again showing the same sort of effect as that obtained for visual acuity. One of the important factors here is spatial summation in the retina due to the convergence of many receptor cells upon a single ganglion cell. This makes for poor spatial discrimination as well as low fusion frequency.

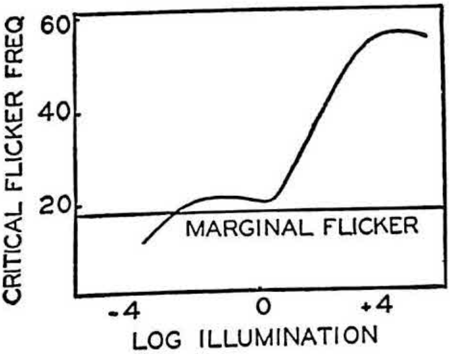


FIG. 14.9. Variation in critical flicker frequency with intensity. Note break in curve representing shift from rod to cone vision. With high intensities, the c.f.f. reaches almost 60. (After Bartley.[9])

Another aspect of temporal patterning in visual perception which has baffled generations of psychophysiologists is referred to as subjective colors. Here a disc, composed of a white ground with groups of narrow lines in deep black drawn upon it, is rotated rather slowly and at the proper speed (depending upon the particular pattern of disc used and upon the individual) one sees, not black lines on white, not gray lines, but colored lines. Distinct hues appear in various parts of the pattern. Figure 14.10 shows one such disc. No acceptable explanation has appeared as to how color can be achieved by the alternation of black and white patterns. If an explanation could be found for this phenomenon, it might unlock some of the secrets of color vision and of the general differentiation of qualities within a single sense.

Temporal patterning becomes the prime object of analysis when attention is turned to electrophysiological recording of action potentials from retina, optic nerve, or visual cortex. Recently much research has had as its objective the analysis of these records. Figures 14.11 through 13 show the appearance of the potentials at several levels within the visual system.[10] Note that the simplest response occurs in the retina; the optic nerve response is more complex and more difficult to analyze; and the cortical response bears little resemblance to that of the retina. Cortical events are, as one would predict, more complicated. But even retinal events are not simple. There are several different kinds of fibers in the retina which respond differently to various temporal patterns of light. Figure 14.11B shows one of the more numerous kinds — an on-off fiber, that is, a fiber which gives a response both when light comes on and when it goes off. In this example, the optic nerve shows a response at the onset of the stimulation and again at the termination of stimulation. Other sorts of fibers are pure "on" fibers, and pure "off" fibers.

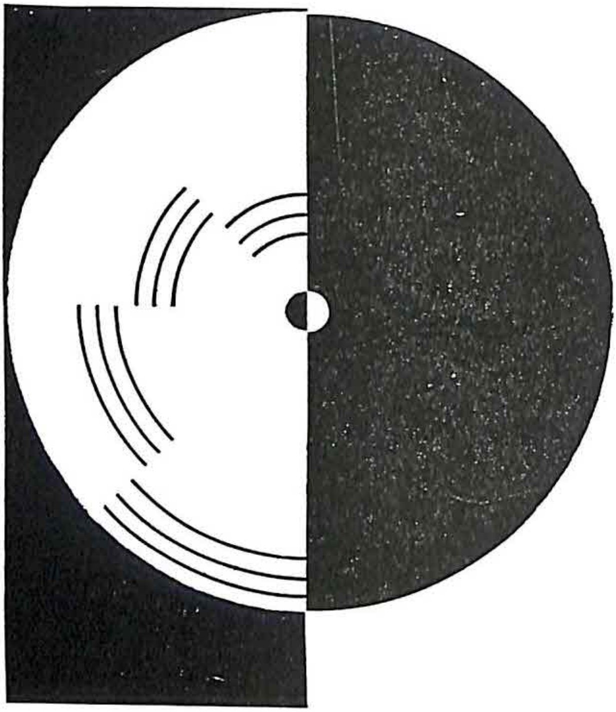


FIG. 14.10. One of the discs used to produce subjective colors. Red appears in the inner ring, blue in the outer, and yellows and greens in the intermediate ones when the disc is rotated in a clockwise direction.

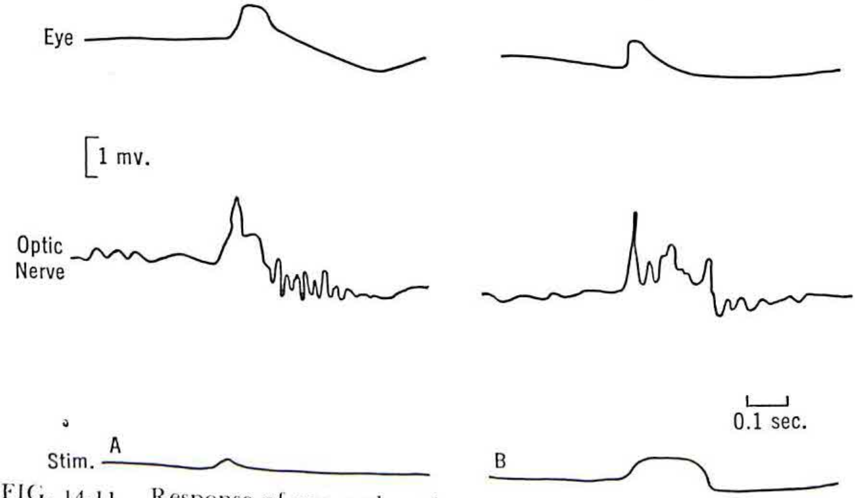


FIG. 14.11. Response of eye and optic nerve of rabbit to white light. Note "off" effect in B. (After Adrian.[10])

These are fixed characteristics, apparently, of some retinal unit, perhaps the ganglion cells. Variable characteristics of retinal action potentials are the latency, frequency, and duration of the burst of impulses. Although the relationships between these and light intensity vary with the type of fiber under discussion, in general, higher intensities give a shorter latent period, greater frequency of impulses, and a greater number of impulses.

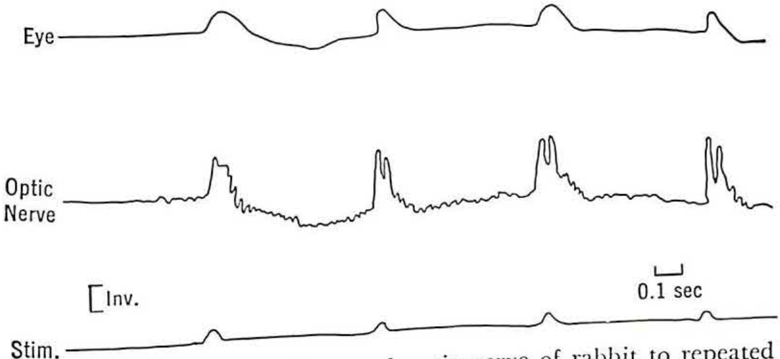


FIG. 14.12. Response of eye and optic nerve of rabbit to repeated flashes of white light. (After Adrian.[10])

Of great interest is a comparison of the electrical (A) and psychophysical (B) data, such as occurs in Figure 14.14. It is important to know whether these essentially neurophysiological data that we have discussed do actually bear upon psychophysiological problems. Is this approach fruitful? This figure tells us that it is. There is close correspondence between the threshold data gathered by two very different methods: (1) by recording of the electrical response of the human retina and (2) by the psychophysical measurement of threshold visual intensities at various wavelengths.

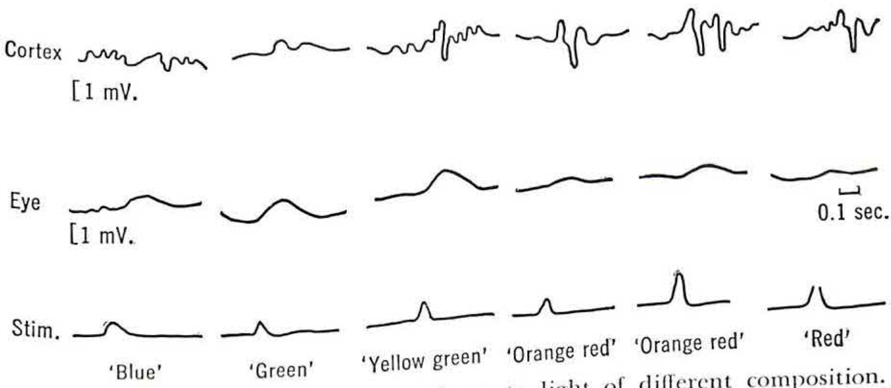


FIG. 14.13. Response of cortex and eye to light of different composition. (After Adrian.[10])

Color Deficiencies. There are many color deficiencies, ranging from total color blindness, the inability to see hue of any sort, to a barely detectable deviation from normal color vision. Usually, color vision is described in three categories (see Table 14-1): **trichromatism,** normal or nearly normal color vision; **dichromatism,** red-green blindness or blue-yellow blindness; and **monochromatism,** or complete hue blindness. There are various subdivisions under each of these. The trichromatic group represents only variations in either the visibility curve or in the relative intensities of the primaries required for matching: all require at least three stimuli for matching all colors. In dichromatism, only two primaries are required. The two forms of dichromatism ordinarily met with are **protanopia** and **deuteranopia,** both forms of red-green blindness. In protanopia, the red end of the spectrum is shortened (as in the scotopic visibility curve) and greens appear as grays. In deuteranopia, the visibility curve is normal, both in extent and peak, but reds and greens are confused with grays, yellowish or bluish as the case may be. In each of these cases, there is a single neutral point (pure gray) which divides the spectrum into two parts of different hue. These two forms, with their counterparts in red-green weakness account for most of the known color deficiencies. The two forms of yellow-blue blindness seem to be very rare, though large and unselected groups of subjects have never been systematically tested with a thorough and reliable test.

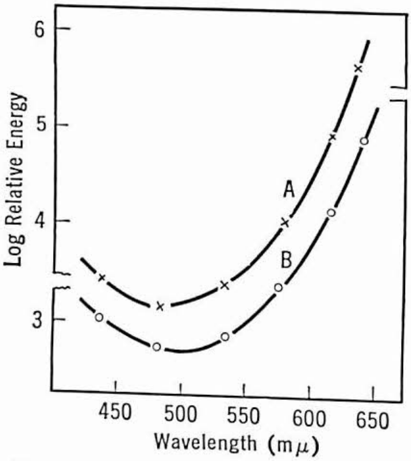


FIG. 14.14. Comparison of the threshold for electrical (A) and threshold for psychophysical (B) data for intensity for light of various wavelengths. (After Riggs and Johnson.[11])

These color deficiencies, largely inherited, are sex-linked characteristics and are found in about 8 percent of males, but only in 0.4 percent of females. Total congenital color blindness, which is rare, does not show these marked sex differences. It is pure rod vision, with foveal blindness, and the inability to see in very bright light. It is possible to acquire color deficiencies of certain types, and even total hue blindness, as a result of accident or disease, but it is not common.

Color vision is tested routinely in certain situations: in the armed forces, for railroad or ship operations jobs, for pilots, and, in general,

Table 14-1. Classification of Types of Color Vision
[After Judd (12)]

Type	*Discriminations*	*Peak* (mμ) *of Visibility Curve*	*Neutral Points* (mμ)	*Name*
Trichromatism	light-dark Y-B R-G	555	None	Normal
	L-D Y-B R-G-weak	540	None	Protanomaly
	L-D Y-B R-G-weak	560	None	Deuteranomaly
	L-D Y-B-weak R-G	560	None	Tritanomaly
Dichromatism	L-D Y-B	540	493	Protanopia
	L-D Y-B	560	497	Deuteranopia
	L-D R-G	560	572	Tritanopia
	L-D R-G	560	470 580	Tetartanopia
Monochromatism	L-D	510	All	Congenital Total Color Blindness
	L-D	560	All	Acquired Total Color Blindness
	L-D	540	All	———

wherever it is important that one be able to identify colored signals without error or hesitation. It is important also for chemists (in qualitative analysis), for biologists, for geologists, for textile and paint and food workers, and in many other occupations. The tests normally used are ones which are simple to use, require no special and complicated situation, and are quick. These are the so-called pseudo-isochromatic charts, of which the best in widespread use in this country is the Ishihara test.[13] The principle upon which these are based is the confusion of hues. The charts contain small dots of slightly different hues and brightnesses. The normal individual sees a number which stands out because of a difference in hue from the background. The color-deficient individual, not seeing the hue pattern, depends upon brightness differences, which are arranged to make a different number, which he reports. These tests are extremely useful for the purposes for which they were designed, but they are not ideal for the scientific investigation of color vision. Until more analytical studies are done on large and random groups of people, the data regarding color deficiencies will probably not throw a great deal of light upon the nature of color vision. At one time there was great hope that the data of color deficiency would enable one to decide conclusively among the theories of color vision. This hope has not been realized.

It is of some interest to know that color vision is not universal in the animal kingdom. It appears sporadically. Fish have color vision, birds and bees do also, but most mammals do not. Dogs and cats are totally color blind, though they often fool one by use of brightness differences. A bull, likewise, is not interested in a red flag — a white one does even better in attracting his attention. But primates, the order to which man belongs, have color vision. Monkeys, chimpanzees, and all the rest of us see hue. The chimpanzee has trichromatic vision almost identical with ours and his visual system is anatomically very similar to that of man.

Theories of Color Vision. During the past 150 years, a large number of theories of color vision have been proposed, none of which is adequate to cover all the known or supposed facts. Current theories can be considered as belonging to three major groups, depending upon the kind of data which are considered of primary importance.

Theories which place first emphasis upon the data obtained from stimulus mixture are often called trichromatic theories. They postulate three kinds of cones, called R (red), G (green), and B (blue) cones, and derive both hue and brightness from the stimulation of the three in various proportions. Each cone type is said to have a broad spectral reponse curve but each has a different peak, so that any narrow band of wavelengths would stimulate all three kinds, but one most strongly. When all three are stimulated equally, the resulting sensation is white (achromatic). These theories stem directly from the Young-Helmholtz theory. The major difficulties are in the explanation of the usual types of dichromatism and in the unfortunate truth that one really cannot match all colors by the mixture of only three primaries without the subterfuge of a negative amount of one of them at some points.

Theories which place prime emphasis upon the psychological data derived from experiments upon after-images, contrast, and the way colors appear to the observer (uniqueness of yellow, for example) are often referred to as four-color theories (although they are really six-color theories). They asume one receptor or one mode of response for each of the six primary colors: R, G, B, Y, Blk., and W; but these receptors or functions are paired so that R and G are opposite in effect and recovery from R stimulation will give the G of the negative after-image. One additional unique feature of such theories is that the achromatic scale is not derived from the same responses as the hue functions. Difficulties arise when one attempts to translate these conceptions into neurophysiology and to specify how these pairings can possibly operate. Here, also, difficulties are encountered with color deficiencies, since not enough latitude is permitted to cover all the known variations.

The third category of theory is derived from the recent neurophysiological evidence of electrical recording from the visual system. These theories postulate a dominator system which accounts for the achromatic series and a number of modulator systems (at least six or seven), responding to somewhat different distributions of wavelengths, which cover the visible spectrum. These modulators are grouped into three main spectral regions, which correspond to some extent with the R, G, and B regions of the trichromatic theory. Extensions of the theory enable one to account for the major classes of color deficiencies. Problems of contrast (simultaneous and successive) are wisely put aside because of the fact that not enough is yet known about the neurophysiology of inhibition or of the various categories of visual units such as " off-systems." Hue discrimination and some of the difficulties of predicting the results of stimulus mixture are well taken care of.

None of these theories can be said to be complete or to explain all the facts, but they are useful in summarizing a number of diverse facts. The character of the information available is beginning to change now that attack is possible upon the elements of the visual system. It is probable that the color theories of the future will not only show great changes from those that have held the field for so long, but also that they will become much more precise in their description of the nature of the events which are to account for the great complexity in both patterning and qualities which occurs in vision.

Suggested Readings

1. Bartley, S. H., *Vision*. New York: Van Nostrand, 1941, pp. xv and 350.
2. Judd, D. B., "Basic Correlates of the Visual Stimulus." Chap. 22 in Stevens, S. S. Ed., *Handbook of Experimental Psychology*. New York: Wiley, 1951.
3. Graham, C. H., *Visual Perception*. Chapter 23 in *ibid.*
4. Bartley, S. H., *The Psychophysiology of Vision*. Chapter 24 in *ibid.*
5. Chapanis, A., *How We See*. Chap. 4 in Chapanis, A., Garner, W. R., and Morgan, C. T., *Applied Experimental Psychology*. New York: Wiley, 1949, pp. 67–117.

15 Secreting Cells and Chemical Integration

The Differentiation of Secreting Cells. One of the basic functions of protoplasm is secretion. These secretory activities of single cells may be thought of as fundamental chemical controls of life processes. It probably is true that most cells of multicellular animals have some secretory functions; that is, some cellular products liberated by them have effects upon other cells, tissues, or organs in the body. But, in the organs which we call **secreting glands,** these chemical activities of cells have become highly specialized. Each of these glands is composed of one or more groups of cells which produce and secrete their own kind of chemical substance or substances. Some of these substances, such as saliva or sweat, are secreted through ducts into a cavity of the body or to its surface. Others which are called **hormones** pass into the blood stream, are carried by the blood to all parts of the body, and have some effect on other parts. It is these hormones from the ductless or **endocrine glands** in which we shall be most interested, for they are very important in an understanding of behavior.

There are three criteria for classifying a given structure as an endocrine gland: (1) A group of cells specialized for secretion, (2) whose secreted chemical substance is carried in the blood stream, and (3) which has an effect on another part of the body. Duct glands, such as the tear or sweat glands or the outer pancreas, satisfy the first and third of these criteria only. We shall study the functions and effects of each of the hormones separately, but it must be kept in mind that their effects usually do not occur separately in the organism. The effect of one usually involves the functioning of others. We have, then, in the endocrine system, another means of integrating the behavior of organisms — a **chemical integration.** Although this chemical integration is largely mediated by the endocrine glands themselves, we shall see that it is aided by the nervous system.

The Endocrine System in Man. Topographical views of the endocrine glands in position in the body are quite common in textbooks. Some such views include the pineal gland within the cerebrum and the thymus gland within the thorax or chest cavity. Although it has been thought by some that these two organs represent true endocrine glands, we shall not include them. Contemporary investigations have afforded no clear evidence that these two bodies have endocrine functions.

Figure 15.1 will provide a quick view of the nine best known endocrine glands in man, but it should be pointed out that this is not a complete picture. For example, the walls of the intestines contain four kinds of endocrine cells that are brought into action by foods; the placenta in pregnant women is known to have very active endocrine functions; some authors speak of a third part of the pituitary gland; and others believe that the liver possesses endocrine functions. However, some understanding of the major functions of the nine glands shown in Figure 15.1 will be sufficient for our present purposes.

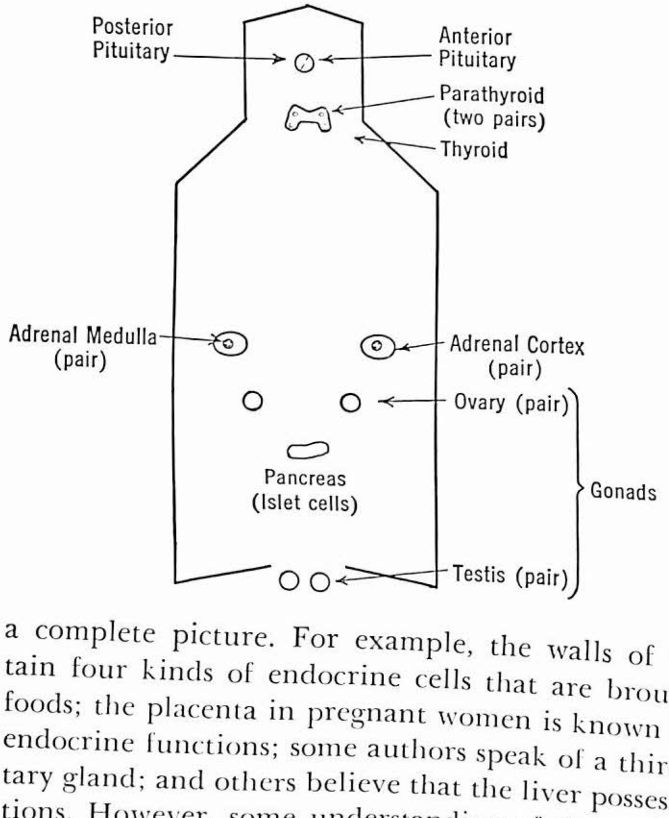


FIG 15.1. The relative positions of the nine major endocrine glands in man (with the pituitary regarded as two different glands). Those named at the right are mainly under chemical control; those at the left are mainly under nervous control; and the islet cells of the pancreas are controlled by both means.

In the following discussion we shall speak of each kind of gland in the singular, whether one, a pair, or more is involved. Note in the figure that the **anterior** and **posterior pituitary** are represented as two separate glands. There are several reasons for this separate treatment. (1) They are different anatomically; (2) the anterior portion has no known nervous control while the posterior portion is under direct control of nerve fibers from the hypothalamus; and (3) the anterior portion controls many other endocrine glands and, in turn, is affected by them. The anterior pituitary, therefore, is sometimes referred to as the " master " gland of the endocrine system, so perhaps you should know its other name, **the adenohypophysis.** The posterior pituitary also is referred to as the **neurohypophysis** because of its intimate relationship with the nervous system; and the whole pituitary organ may be called the **hypophysis.** Note also that one other gland, the **adrenal medulla,** is largely under nervous control; and that the inner portion (**islet cells**) of the pancreas is activated by both chemical and neural stimulation. The **thyroid** has a nerve supply to its blood vessels, but nervous stimulation is not a major factor in its function. Instead, the thyroid, the **adrenal cortex,** and the **ovary** and **testis (gonads)** are stimulated to secretion by the hormones of the anterior pituitary. Let us now consider the functions of each of the hormones secreted by these nine glands and try to gain some appreciation of their role in the chemical integration of human behavior.

MAJOR FUNCTIONS OF THE HORMONES

The major functions of the principal hormones of the nine endocrine glands mentioned in the preceding section are shown in Table 15-1. The table is necessarily incomplete and is to be considered only as a tool for facilitating an over-all view or review of the material contained in this chapter.

Thyroxin: Thyroid Gland. The thyroid gland, which lies on either side of the larynx in the neck in two attached lobes, is sometimes called the " pacemaker " of the body. This designation is quite appropriate, for the amount of its hormone, **thyroxin,** controls in large measure the rate at which our life processes function; that is, it controls the rate of metabolism. Any measure of metabolic rate then could be construed as reflecting indirectly the rate of thyroid secretion. The single test most frequently used for this purpose, a measure of the rate of oxygen consumption under fasting and resting conditions, is known as the **basal metabolism test.**

With large amounts of thyroxin, the rate of metabolism is increased, more oxygen is consumed because we are " burning fuel " more rapidly, and many activities of the body are speeded up. The blood pressure and heart rate are increased, the body produces more heat, and we become more active muscularly, show more enthusiasm and drive, and seem to have more energy; but we experience difficulty in relaxing and in sleeping. Since food-stuffs are consumed so rapidly, we do not gain weight; and we may even lose weight, as stored fats are utilized. Mental activity also is somewhat speeded but

Table 15-1. Principal Hormones and Their Major Functions

Gland	Hormone	Major Functions
Anterior pituitary	Thyrotrophic (TTH)	Stimulates thyroid secretion
	Adrenocorticotrophic (ACTH)	Stimulates secretion of some hormones of adrenal cortex
	Lactogenic (prolactin)	Stimulates milk secretion by mammary glands
	Luteinizing (LH)	Development of interstitial cells of testis and ovary, and corpus luteum
	Follicle-stimulating (FSH)	Development of spermatogenic tissue in male and follicle in female
	Growth (STH)	Stimulates growth
Posterior pituitary	Oxytocin	Excites nonstriated muscles especially of uterus; excites mammary glands
	Vasopressin	Produces rise in blood pressure
	Antidiuretic	Prevents loss of water through kidney
Thyroid	Thyroxin	Influences metabolic rate
Parathyroid	Parathormone	Maintains calcium and phosphorous balance in blood
Islet cells	Insulin	Necessary for utilization of blood sugar
Adrenal cortex	Cortical Steroids	Increased carbohydrate metabolism, sodium retention and potassium loss; some androgenic and estrogenic effects
Adrenal medulla	Epinephrine Nor-epinephrine	Increased sugar output by liver, stimulate most SNS end-organs (differentially)
Ovary	Estrogen	Produces female primary and secondary sex characteristics
	Progesterone	Prepares uterus for implantation of embryo
Testis	Androgen (Testosterone)	Sexual arousal; produces primary and secondary sex characteristics

not necessarily improved and, as the amount of thyroxin is increased, it soon becomes sporadic (possibly due to muscular restlessness) and sustained mental work becomes difficult or impossible. Extreme emotional behavior is typical. This pattern of behavior, known as **hyperthyroidism,** is a fairly common disorder. It may be controlled by removing some of the gland or by administering a thyroid-inhibiting drug.

Many people are in need of additional thyroxin, however, and some of the properties of the hormone can best be understood from a description of some of the symptoms of severe **hypothyroidism,** of which there are at least two classes: **cretinism,** in which there is deficient thyroid function at birth or during the early years of life; and **myxedema,** in which the deficiency occurs in adults. Now that thyroxin is available commercially, cretinism is rapidly disappearing. Without supplementary thyroxin, however, the cretinous child fails to develop in a normal manner physically or mentally. If he lives, he retains a dwarfed stature and a severe mental deficiency, since thyroxin is necessary for normal calcification of bones and normal mental development. Later medication cannot help him.

Myxedema, however, sometimes occurs in a person who has enjoyed more or less normal thyroid function and it easily may be eliminated by additional thyroxin. Its major symptoms are the opposite of those described for hyperthyroidism. Such a person usually is sleepy, sluggish mentally and physically, unemotional, lacking in enthusiasm, and may gain weight. Moreover, his skin and hair become excessively dry and the hair becomes brittle and breaks easily, thus producing a kind of baldness. It is apparent that the effects of the thyroid hormone are wide-spread and important both in medicine and in psychology.

Parathormone: Parathyroid. The major function of the hormone of the **parathyroid glands,** sometimes called **parathormone,** is the regulation of calcium metabolism, although it serves also to regulate the metabolism of phosphates. The glands, which usually are four in number in man, are quite small and two are imbedded in or attached to each lobe of the thyroid gland (hence the name, " parathyroid "). In the absence of parathormone, calcium quickly becomes depleted from the blood. Since calcium is necessary for normal nervous and muscular activity, neuromuscular processes quickly become abnormal. Muscle twitches **(clonus)** occur, the muscles become weakened, and ultimately the respiratory muscles fail. The condition can be compensated for by the injection of calcium solution into the blood, but as soon as the calcium has been utilized the deficiency symptoms reappear. Thus, it is obvious that parathormone is necessary not only for normal nervous and muscular activity but for life itself. Fortunately, dysfunction of this gland is rare. An adequate secretion of parathormone seems to be stimulated directly by low levels of blood calcium.

Cortical Steroids: Adrenal Cortex. The **adrenal glands** are a pair of small bodies, each of which is embedded in the top surface of the kidney (hence the name, "adrenal"). Each has two independent secreting parts: (1) the center or **adrenal medulla,** and (2) the outer covering or **adrenal cortex.** A number of separate substances are secreted by the cortex which are known as **cortical steroids,** but sometimes the total secretion is called **cortin.** The functions of these steroids are very complex and furnish a vital topic of research today. It is known that they serve to regulate the metabolism of sodium chloride and other salts, of water, and of carbohydrates; and that they are necessary if the organism is to be able to withstand periods of stress. It is also known that some of the steroids are like or similar to the gonadal secretions; and excessive secretion of cortin, due to a tumor or some other factor, may cause excessive hair growth and other masculine signs in a woman, or may interfere with normal adult development in either sex. Deficiencies over a period of time result in emaciation and dehydration of the body, with extreme fatigue and weakness. Temporary deficiencies are just beginning to be appreciated and are receiving much attention, especially in relation to studies of physiological stress.

Epinephrine: Adrenal medulla. The medulla of each adrenal gland is practically surrounded by its respective cortex. It is an unusual endocrine gland in several respects: (1) of all the endocrines it is perhaps the least essential to life, yet it prepares the organism to meet emergencies; (2) it is innervated by the sympathetic nervous system; and (3) it facilitates the activity of most of the other end-organs stimulated by the SNS. Its hormone, **epinephrine,** (sometimes called **adrenalin**) will largely mimic the effect of SNS stimulation.

The function of epinephrine will be discussed in considerable detail in connection with Chapters 17 and 21. In this chapter, we shall emphasize its relation to blood sugar; but note that it is a powerful stimulant for the increase of heart rate and blood pressure, and for the dilation of the passages in the lungs — the **bronchia.** Thus, a commercial preparation of the hormone is a standard emergency medicine in every physician's kit. Its action on the heart in cases of shock, and its action on the bronchia in acute asthmatic crises, has saved many lives.

Epinephrine promptly acts to raise the level of blood sugar by stimulating the liver to convert **glycogen** into **glucose** and then to release it into the blood stream. A low blood sugar level is itself an adequate stimulus for this chain of reactions, but the typical route of stimulation is via the SNS which quickly responds to emergency situations. Regardless of how the stimulation occurs, however, epinephrine operates to raise the available blood sugar. The action of some of the cortical steroids support it in this function.

It has been demonstrated recently that the adrenal medulla actually secretes two slightly different forms of its hormone. The second form, known as **nor-epinephrine,** is regarded as an uncompleted epinephrine. It may prove of real importance in physiological psychology, but further comments about it must await the results of current research.

Insulin: Islet Cells of Pancreas. Although the outer portion of the pancreas is a duct gland, its center portion is composed of groups or islands of cells which have only endocrine functions. Their hormone is **insulin,** the discovery and commercial production of which has made it possible for persons afflicted with insulin deficiency (**diabetes mellitus**) to lead normal lives. Insulin makes possible the utilization of blood sugar by the cells of the body. In its absence, sugar cannot be used by the cells even though the blood-sugar level is high. Since the nervous system in particular needs sugar for normal function but cannot store it, a decrease in insulin in the blood soon will have disastrous results. They are known as **diabetic coma** and will soon lead to death unless insulin is injected. Too much insulin also will result in unconsciousness and, ultimately, death; but here the mechanism is somewhat different. A high blood-insulin level results in rapid utilization of available blood sugar and, although insulin also stimulates the liver to prepare additional stores of glycogen, the process is not immediate. When the immediate supply is exhausted the nervous system begins to fail and only an addition of glucose to the body can arrest the process. This is known as **insulin shock.** It sometimes is used in the treatment of schizophrenia and will be discussed later in the text.

Although the secretion of insulin apparently is stimulated directly by a high blood-sugar level, it is stimulated also via cranial nerve X, the vagus.[1] Furthermore, insulin, in antagonism to epinephrine, seems to have the property of facilitating the action of parasympathetic nervous system end-organs. More will be said concerning this characteristic in Chapter 17. It is enough to know now that hyperinsulinism mimics in large part an extensive stimulation of the PNS [2]; and to know that insulin, cortin, and epinephrine together demonstrate a chemical integration that regulates the supply of carbohydrates upon which our every action depends.

Hormones of the Posterior Pituitary. Although three hormones are listed for the neurohypophysis in Table 15-1, there is clear evidence for only two, **oxytocin** and **vasopressin;** and of the two the latter seems the more important in day-to-day life.[3] We have included all three in our treatment, however, since many investigators believe that some day the **antidiuretic hormone** will prove to be separate from the vaso-pressor hormone. At the moment, the hormones of the neurohypophysis are of little direct significance for psychology. They are included to give you an example of an endocrine gland which is solely under nervous control and to give you a more complete picture of pituitary function.

Oxytocin. Although oxytocin has some effect on most smooth muscles of the body, its major effect is on the muscles of the uterus. It is believed to be responsible for the extraordinarily strong contractions at the end of pregnancy which constitute "labor," and which result in the delivery of the infant from the uterus, and it is known to have strong stimulating effects upon milk secretion by the mammary glands.

Vasopressin. The major control of blood pressure seems to be via the autonomic nervous system, and the significance of vasopressin is not well understood. Removal of the neurohypophysis, however, results in low blood pressure, and increases in blood pressure follow injection of posterior pituitary extracts.

Antidiuretic Hormone. There is no doubt that an antidiuretic hormone is largely responsible for the control of water retention or excretion in our bodies. Hyposecretion of the hormone leads to a disorder in which there is more or less constant urination with, of course, great thirst. In other words, water is not resorbed in the kidneys but passes to the bladder and is excreted. Some workers believe that it is vasopressin, and not a separate hormone, which has these antidiuretic effects.

Hormones of the Anterior Pituitary. The hormones of the adenohypophysis are complex and extremely interesting. They are secreted apparently by only three types of cells, yet some authors are convinced there are at least nine separate hormones and others suggest even more. We shall content ourselves with the study of the six hormones best known and most clearly differentiated. It is possible that one of them, the **growth hormone,** is responsible for many of the effects sometimes ascribed to additional postulated hormones.

Two of the six (the growth and **lactogenic hormones**) act directly on nonendocrine tissues; the other four act primarily on other endocrine glands and induce hormone production and secretion in them. These four are known as **trophic hormones;** that is, each has a "target" endocrine gland which it induces to activity. It is probable, however, that all six of these hormones have endocrinotrophic functions; it is by virtue of these trophic hormones that the anterior pituitary gains the name "master gland."

Growth Hormone (STH). Pituitary dwarfism and gigantism are two of the dramatic effects of glandular dysfunction. In cases of hyposecretion of the growth hormone (sometimes referred to as the somatotrophic hormone, or *STH*)[4] from the anterior pituitary, we find an inhibition of growth in the tissues and the long bones of the body, resulting in a shortness of stature known as dwarfism. Such individuals may be normal mentally and may or may not have other malformations in structural development. The salient feature is the marked arrest in growth. On the other hand, hyperfunction of this portion of the gland during the years of normal growth will result in an extended growth period and occasionally will produce giants 8 or 9 feet in height. Again, the mental development may be normal and there may be no malformation in other bodily structures.

Hyperfunction in this gland in the post-pubescent period results in another kind of anomaly, **acromegaly.** Here, by reason of a tumorous growth or some other unusual stimulation of the gland after the period of normal growth has been terminated, a new period of growth begins. At this time, however, the ends of the long bones have typically become rounded and closed and this interferes with further longitudinal growth. As a result, the increased calcification that occurs under this renewed pituitary stimulation results in a widening of the bone structures; and the increased tissue growth must occur in relation to a fixed skeleton — that is, in the organs of the abdomen and in exterior skeletal muscles. This kind of growth is particularly noticeable in the extremities of the skeleton and in the viscera. Thus, the abdomen, hands, feet and facial structures acquire a heavier and coarser appearance.

The Lactogenic Hormone. As its name implies, the **lactogenic hormone** (sometimes called **prolactin**) stimulates the secretion of milk in the mammary glands. Its secretion is believed to be increased by endocrine changes that occur late in pregnancy or at the time of delivery; but other hormones already have prepared the mammaries. The secretion of prolactin is merely the final step in the process.

Thyrotrophin (TTH). Of the trophic hormones of the anterior pituitary, **thyrotrophin,** or **TTH,** is perhaps the most specific in its effect. Apparently its sole function is to stimulate the thyroid to secrete thyroxin. In its absence, the thyroid is practically nonfunctional. If there is over-production of thyroxin, however, the production of TTH is inhibited. The joint actions of TTH and thyroxin furnish a neat example of chemical integration. The major principle is shown in Figure 15.2. Normal thyroid secretion, then, is the result of a nice balance between two hormones which is achieved through their mutual chemical influences.

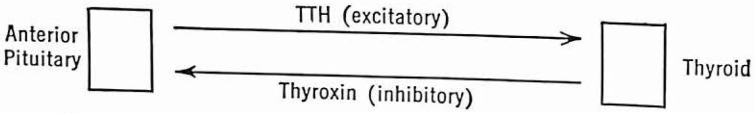


FIG. 15.2. Chemical integration in thyroid secretion.

Adrenocorticotrophin (ACTH). The major function of **adrenocorticotrophin,** or **ACTH,** is to stimulate the adrenal cortex. It is the subject of vigorous research at the moment. Its discovery and isolation is only recent, but it is being heralded as a panacea for all sorts of disorders, from rheumatoid arthritis to allergic asthma. Its functions, apart from its stimulation of the adrenal cortex, are not clear, but it seems to have some influence upon the formation of acetylcholine. It apparently is released in increased quantities during stress stimulation and is important in withstanding prolonged stress. We shall refer to it again when we discuss the " general adaptation syndrome " in Chapter 25.

The Gonadotrophic Hormones. The two **gonadotrophic hormones** of the anterior pituitary primarily affect the gonads; that is, the ovaries and the testes. They, with the gonadal secretions, are largely responsible for sexual maturation and function, and for regulating the menstrual cycle and the reproductive cycle in women. Their functions will be more easily understood if we discuss them in relation to the gonadal hormones themselves. They are known as the **follicle-stimulating hormone (FSH),** and the **luteinizing hormone (LH).**

The Gonadal Hormones. In a developing vertebrate organism, the undifferentiated pair of gonads in the abdomen undergoes differentiation according to the sex of the organism. In the female, they develop into ovaries; in the male, they descend into a sack (the scrotum) outside the body and are called testes. Under the influence of the gonadotrophic hormones from the anterior pituitary their tissues mature, they produce germ cells called **ova** in the female and

spermatozoa in the male, and they also become endocrine glands. Both secrete several substances, all of which are chemically similar to the steroids of the adrenal cortex. Those primarily from the testis are known as **androgens** and those primarily from the ovary are known as **estrogens;** but the testis apparently produces some estrogen and the ovary some androgen, and some of both also is produced by the adrenal cortex. Moreover, during the period of reproductivity in a female, the ovary at times secretes an additional hormone, **progesterone.** Since the gonadotrophic hormones were discovered and named in connection with studies of female physiology, we shall first discuss the female hormones.

Estrogen: Follicles of Ovary. The cells that secrete estrogen in the female are in the walls of the **follicles,** small bodies within the ovary most of which contain a developing egg cell or ovum. Estrogen secretion is stimulated by a gonadotrophin from the anterior pituitary, the follicle-stimulating hormone (FSH). Under the influence of FSH and estrogen the girl becomes a woman, her sexual organs mature, her mammary glands grow, and she attains a typical female body build and hair distribution.

In the sexually mature woman, usually only one ovum attains maturity each 28 days or so. At that time it breaks through the follicle wall and the covering of the ovary (a process known as **ovulation**) and begins its descent through tubes leading to the uterus.

Progesterone: Luteal Body of Ovary. Just before and after the rupture of the follicle at ovulation, other cells in the follicles have been undergoing development under the influence of FSH and the other pituitary gonadotrophin, the luteinizing hormone (LH). These cells together are known as the luteal body or the **corpus luteum.** Soon, under the additional influence of prolactin from the pituitary, they are led to produce and to secrete another hormone, **progesterone.** Its name well describes its major functions; it augments the preparation of the body for gestation, for the period of development of the fertilized ovum into a human infant. With estrogen, it prepares the uterus to receive and implant the ovum, and it maintains the change begun under estrogen in the character of the uterine wall, where new vascular tissue has been added and, therefore, an enriched blood supply has occurred.

If the ovum has been fertilized and if it becomes implanted in the uterus, the corpus luteum continues to secrete progesterone for awhile, but soon a new chemical influence becomes predominant in the maintenance of pregnancy, the **chorionic hormone.** This product, peculiar to pregnant mammals, is produced by endocrine tissue in the placenta and is similar in many respects to progesterone. Its inhibitory influence on the production of FSH is removed, of course, immediately after birth and the normal reproductive cycle soon reappears, although in some species its reappearance is somewhat retarded if the newborn infant is nursed. The mechanism of this further inhibition is not well understood.

If pregnancy has not occurred, the corpus luteum degenerates, progesterone secretion decreases rather abruptly, and menstruation oc-

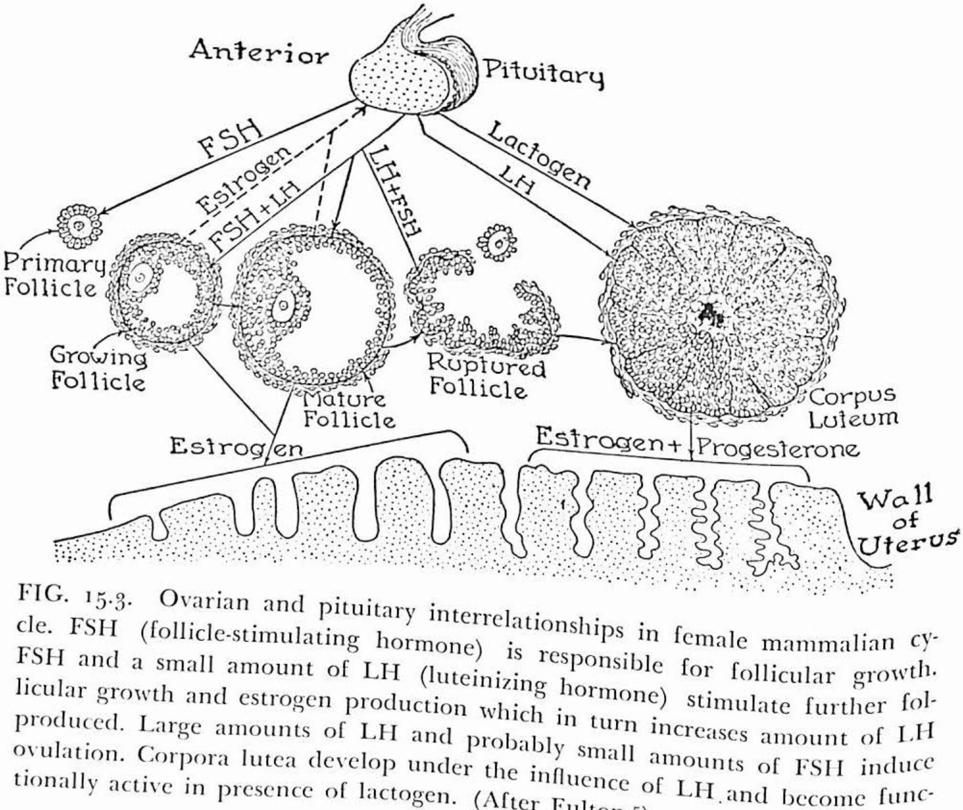


FIG. 15.3. Ovarian and pituitary interrelationships in female mammalian cycle. FSH (follicle-stimulating hormone) is responsible for follicular growth. FSH and a small amount of LH (luteinizing hormone) stimulate further follicular growth and estrogen production which in turn increases amount of LH produced. Large amounts of LH and probably small amounts of FSH induce ovulation. Corpora lutea develop under the influence of LH and become functionally active in presence of lactogen. (After Fulton.[5])

curs, with a disruption in the uterus of the progestational changes, and the reproductive cycle begins anew. These events are quite complex and all of the hormonal relationships are not definitely known, but Figure 15.3 will give you some appreciation of them. Together, they furnish a dramatic example of chemical integration.

Androgen: Testis. The pituitary gonadotrophins also regulate sexual functions in the male. FSH influences the development of the cells of the testis that produce spermatozoa; while LH stimulates and regulates the secretion of androgen from other cells in the testis.

The name of the major androgen is **testosterone.** Under its influence a boy develops into a man, his sexual organs ondary sexual characteristics appear (i.e., masculine hair growth, change in voice, masculine skeleton, and muscle formation), and sexual activities increase. If castration occurs before the culmination of this developmental process (that is, before puberty), he fails to show these male characteristics. Moreover, he tends to be obese with a somewhat feminine appearance and to be unaggressive. Perhaps you have seen pictures of such a person, who is known as a eunuch.

Changes produced by castration after puberty are not so dramatic. In fact, sexual activities may remain unimpaired and sterility may be the only perceptible result. Often, however, there is a decrease in energy similar to that experienced in old age, and a decrease in sexual and other motivations. Injections of testosterone will usually reverse this loss of energy and motivation.

Androgen must be thought of, then, as more than a sexual hormone. It is a powerful general metabolic stimulant and its relation to sex drive and to energy output is of particular interest to psychologists. For example, one experiment in which individual differences in androgen secretion in adolescent males were correlated with measures of aggression concluded that some aggressive behavior is influenced by level of androgen secretion.[6]

Endocrine Functions and Behavior. It is difficult to prepare a relatively simple account of endocrine functions and still keep it reasonably accurate and complete. Much must be left unsaid. Many entire texts, of course, deal with **endocrinology** alone, and there is one major volume called *Hormones and Behavior*.[7] Such a wealth of material cannot be presented in a single chapter of an introductory text. However, we want to re-emphasize the relationship of the hormones to behavior.

The relationships already mentioned involve the following forms of behavior, each of which is important in a study of psychology: level of muscular activity, energy level, insomnia, general motivation, sexual drive, emotion, intellectual ability, masculinity and femininity, aggressiveness, enthusiasm, normality of growth and sexual functions, normality of CNS and ANS functions, and ability to respond to emergency situations and to withstand prolonged stress. Moreover, one should consider the anomalies in physical development or function which are attributable to endocrine dysfunctions, for they also contribute to individual differences in behavior and personality. Finally, the role of the gonadal and gonadotrophic hormones in the determination of reproductive and maternal activity is a major example of the importance of hormones and chemical integration in any study of behavior. The subject will receive further attention in Chapters 20, 21, and 24.

The extent to which individual differences in endocrine excretions contribute to personality differences in individuals who show no frank endocrine disorder remains a question. Some investigations suggest that they play an important role, and many authors in the past have written as though all personality differences were due to our endocrine glands. Such an extreme position certainly is in error. But slight differences in hormonal blood levels may well account for differences in temperament, which often is defined as the physiological basis of personality.

Research on temperament, however, has not been extensive. This is not due to lack of interest, for the subject has occupied the attention of writers since the time of the early Greeks. It is due, instead, to lack of adequate research tools — in particular, techniques for measuring slight physiological differences. When precise and practical techniques are at hand for measuring slight differences in blood hormone levels, some fascinating research can be undertaken. Today such methods are crude and laborious, and few psychophysiologists have used them. Our knowledge is, therefore, scant.

We do have some insights, however. Most is known about thyroid effects, and here there is little doubt that slight deficiencies or excesses serve to alter personality. Next in certainty are the behavioral changes that occur in female mammals associated with known changes in hormone levels during the menstrual cycle; and testerone level in males seems to bear some relation to sexual drive, aggressiveness, and over-all energy level. Beyond this we can only guess that here lies a rich field for research.

Suggested Readings

1. Beach, Frank A., *Hormones and Behavior*. New York: P. B. Hoeber, 1949.
2. Hoskins, Roy G., *Endocrinology*. New York: Norton, 1950.

16 | *Reflexive Behavior*

Although as students of psychology we are interested primarily in the activity of the total organism, a consideration of the relatively simple and direct stimulus-response patterns, called reflexes, cannot be omitted. Not only do they enter into all of our bodily activities, but also life itself depends upon the proper operation of such vital reflexes as breathing. Furthermore, the first genuinely systematic studies of the neural events underlying movement were done upon reflex activity and so a large body of knowledge concerning reflexes is available to give us clues as to how movement is organized.

Reflex Defined. A **reflex** may conveniently be defined as a relatively simple, innate response to a discrete stimulus. No doubt you are already familiar with some examples. Very easy to demonstrate is the **pupillary reflex,** which you may observe in a mirror by shining the beam from a flashlight into your eye. Another example is the knee-jerk, or **patellar tendon reflex,** which the physician often tests during a physical examination. Perhaps less familiar are the " jerks " of other muscles, such as those lifting the lower jaw, and the autonomic reflexes maintaining the blood pressure.

As we have previously pointed out in the discussion of encephalization, reflexes are normally under the influence of events occurring elsewhere in the nervous system. In fact, in order to study a " pure reflex " the physiologist must resort to operative techniques to isolate the mechanism which he wishes to study. Perhaps you have observed that the physician has difficulty in arousing a good knee jerk when you are paying attention to his hammer, but when you follow his instruction to clasp your hands together and pull, a vigorous knee jerk appears. This effect of clenching the hands upon the strength of the knee jerk has been studied systematically and the extent of the leg swing has been measured for different strengths of clench. The results clearly demonstrate that the knee jerk is by no means independent of events elsewhere in the organism.

Most of the things which we shall have to say in this chapter about reflexes will be based, as is implied above, upon studies of isolated mechanisms. Spinal reflexes, for example, have been most extensively studied in preparations in which the spinal cord has been isolated from the rest of the central nervous system. Postural reflexes of, say, the midbrain have been studied by removing the higher central nervous structures. Therefore, our previous discussion of the technique of ablation (Chap. 4) applies with direct force to investigation of such isolated reflexes. Keeping this point in mind, we shall now proceed to the consideration of some of the more important somatic reflexes. Autonomic or visceral reflexes will be discussed in the next chapter.

SPINAL REFLEXES

The **spinal reflexes** have been very extensively studied by physiologists and they serve, therefore, as a convenient starting point for our discussion. The spinal cord is very richly supplied with interconnections, not only directly between afferent and efferent neurons, but also through more or less complex systems of association neurons. In fact, some reflexes, the **scratch reflex** for example, can involve almost the entire organism, even when the spinal cord is completely isolated from the brain. In the intact animal, the spinal cord has important conduction functions, which are discussed briefly in Chapter 3 and in the chapters concerning somesthesis and motor control. As a result, neurons representing influences from every level of the CNS play upon the **final common path.** The diagram (Fig. 16.1) of a single lower motor neuron gives some idea of the tremendous number of synapses on just one cell. It is apparent, then, that what happens in the final common path must somehow depend upon the combined activity of the presynaptic neurons, some of them excitatory, some perhaps inhibitory. In the intact animal, therefore, reflexes are altered by influences from higher centers and, by the same token, influences from higher centers are altered by the reflex activity of the cord. It is not easy to walk without limping if you have a sharp nail in your shoe! The tack stimulates pain receptors which leads to a **flexion reflex.** This will be the first reflex which we shall consider since it serves as a very convenient example of a spinal reflex. In the course of our discussion, we shall have occasion to introduce several concepts of reflex action in general.

The Flexion Reflex. It seems highly probable that men must have noticed the flexion reflex many years ago. The elevated position of the injured limb of a four-legged animal is so universal that it could not have escaped the notice of ancient hunters and horsemen. This flexion of an injured limb is easy to demonstrate in the **spinal animal** and has been very extensively studied. In discussing it, we shall find occasion to introduce a few terms general to reflex activity.

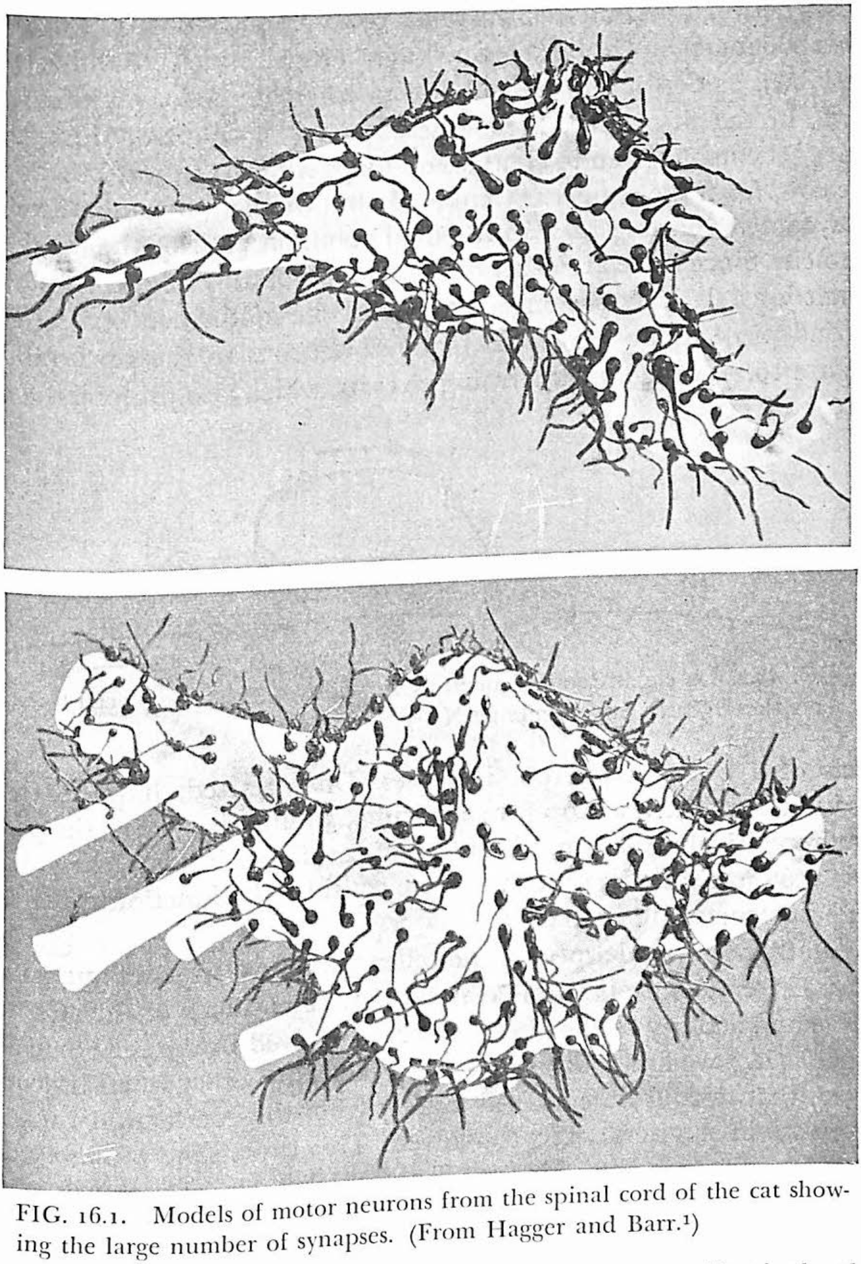


FIG. 16.1. Models of motor neurons from the spinal cord of the cat showing the large number of synapses. (From Hagger and Barr.[1])

Although stepping on a tack will demonstrate qualitatively the flexion reflex, and the raising of a shocked leg is often used as the unconditioned reflex in learning, careful study requires the isolation of a single flexor muscle and the electrical stimulation of single nerve trunks. In such a preparation, stimulation will give rise to contraction of the isolated muscle. If we compare the contraction aroused by a single strong shock to afferent neurons to that aroused by shocking motor neurons, we find that the reflex contraction is more prolonged. This phenomenon has been termed the " after discharge " and is best explained as depending on widespread interconnections within the cord. In the first place, these interconnections involve " long circuits," the name given to conduction loops running upward through the cord from the segment stimulated and, then, after some number of synapses, running back to the final common path of the original segment. Since there are many of these loops, of differing lengths, the result is a relatively prolonged arousal of the motor neurons. In the second place, there are closed loops of neurons in the cord (as in Figure 16.2), called **reverberating circuits,** which also prolong the effects of a burst of afferent impulses. Once started, impulses will travel around such a loop for some time, stimulating the main pathway repetitively. Such circuits, often of considerable complexity, will come to our attention again when we consider the functioning of the highest structures of the CNS.

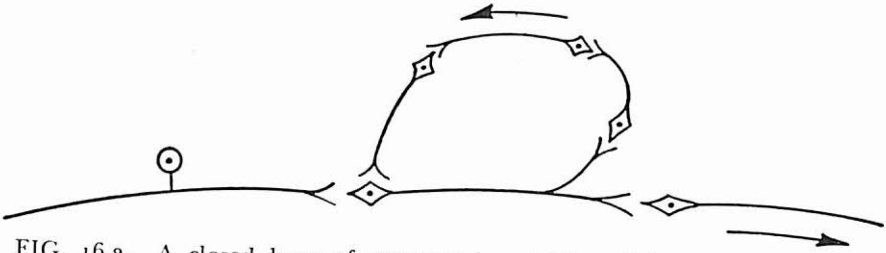


FIG. 16.2. A closed loop of neurons in which activity may persist for some time. (Based on Lorente de Nó.[2])

Another temporal aspect of the reflex will also command our attention. If we measure the time elapsed between a shock to the afferent nerve and activity in the efferent nerve involved in the reflex we are considering, we find that this time is greater, by perhaps a millisecond or so, than would be expected merely from the conduction time of impulses in the nerve fibers involved. This extra time probably depends on three factors. First is **synaptic delay,** which is the time required for an impulse to be effective across a synapse. This time is very short — indeed, it may be vanishingly small with improved measurement. Second, the endings of the afferent fibers are very tortuous. The neural pathways are thus actually longer than appears at first glance and, also, conduction speed is undoubtedly slower near the endings. Third is a more complicated factor called **nuclear delay.** To understand it, we must first note that most reflexes involve more than an afferent and efferent neuron, synapsing in some nerve center. Although the simplest **reflex arc** need contain only receptor, afferent neuron, efferent neuron, and effector in that order, such is the case only for the stretch reflex (cf. Chapters 5 and 9). In other cases, there will be some number of association neurons interposed. Now let us consider the diagram in Figure 16.3. Here the afferent neurons discharge into an intermediate " pool " of complexly interconnected association neurons, which in turn discharge into a pool of motor (efferent) neurons. Now, if a simultaneous burst of impulses reaches the association pool, the association neurons will not discharge a simple simultaneous burst of impulses because of interconnections. If they did, the delay in passing the next set of synapses (to the motor neurons) would be no greater than the synaptic delay. Since they do not discharge simultaneously, there will be, as is shown by experiment, a delay greater than the synaptic delay — this is the nuclear delay. In some cases, it may be several times the synaptic delay.

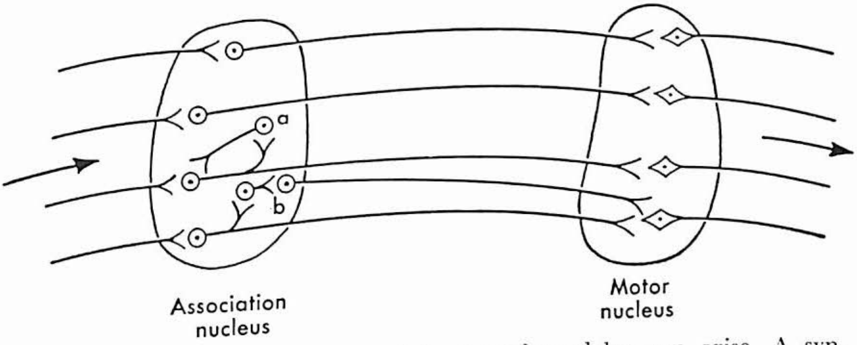
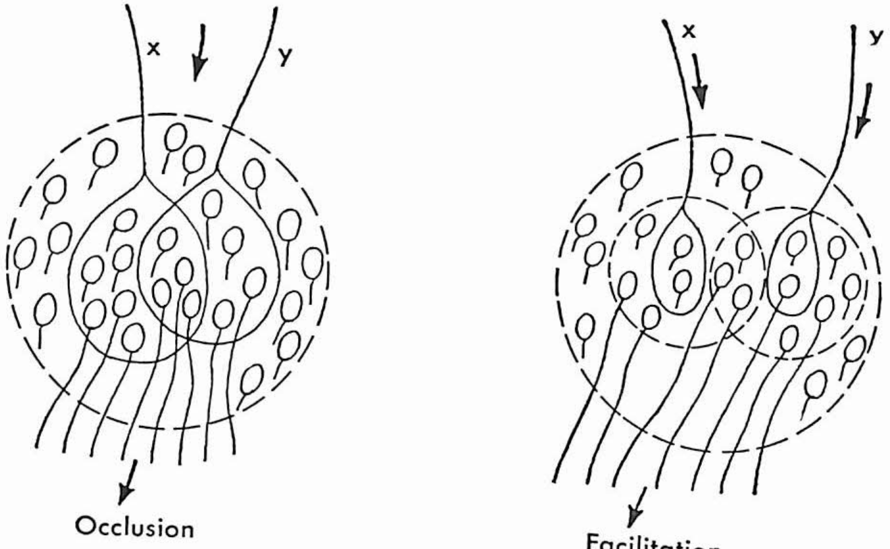


FIG. 16.3. Simplified diagram of how nuclear delay can arise. A synchronous burst of impulses to the association nucleus will result in an asynchronous burst leaving it. Note that a is a closed circuit, b is a parallel arrangement.

It may appear that the rather simple reflex with which we began has developed considerable complication with closer scrutiny. And we are not entirely finished with it, since two other general concepts require consideration. The first of these is designated by the term **occlusion.** The experimental basis of this concept is as follows. Suppose we stimulate three different afferent nerves — say x, y, and z, — and observe the strength with which the flexor muscle under consideration contracts in each case. For simplicity, let us suppose that stimulation of nerve x leads to a contraction of 100 gm., of y to 150 gm., and of z, to 200 gm. What will be the result of stimulating x and y at once? Or of x, y, and z all at once? One might expect addition to occur, so that $x + y = 250$ gm., or $x + y + z = 400$ gm., but such is not the case. In each case, the simultaneous stimulation of more than one afferent nerve leads to a strength of contraction which is *less* than the sum of the contractions taken one at a time. The anatomical explanation lies in the overlapping fields of the central connections of the afferent nerves. Figure 16.4 shows this diagrammatically and it can be seen that if *x* and *y* share some postsynaptic neurons, stimulating *y* in addition to *x* will increase the number of aroused neurons only by the number of unshared neurons.



Occlusion

Facilitation

FIG. 16.4. Illustrating the way in which occlusion or facilitation can arise due to overlapping of fields of influence. In the case of occlusion, the jointly influenced neurons are already activated by n and so y has no further effect on them (the reverse is also true). In facilitation, summation occurs between x and y because their areas of subthreshold influence overlap.

The second concept is designated by the term, **summation,** which may be **temporal** or **spatial.** The concept of temporal summation is derived from the observation that the second of two weak afferent volleys entering the reflex center in rapid succession will have an effect larger than when it is applied alone. The conceptual similarity to temporal summation in the nerve fiber is apparent. Spatial summation is of considerably greater importance in understanding the activity of the CNS (you may recall the statement in Chapter 3 that single impulses probably do not cross synapses). The experimental observation basic here is the exact opposite of that involved in occlusion. If the volley of afferent impulses is too weak to arouse a reflex response, a simultaneous weak volley in another nerve, or in other fibers of the same nerve, can summate in the CNS to give rise to the response. Here one can assume that the one volley of impulses was not sufficient in number to arouse postsynaptic neurons, but the two volleys arriving simultaneously have additive effects in the region in which they have overlapping synaptic connections. When one considers the multitude of synapses on a single neuron (Fig. 16.1), it becomes apparent that in the usual course of events a bewildering complexity of summations (and probably inhibitions) must be constantly occurring.

The Stretch Reflex. The stretch or myotatic reflex has already been discussed in some detail (Chaps. 5 and 9.). It is included here partly because of its importance in the maintenance of posture and partly because it exhibits certain interesting characteristics. Of interest is its so-called " dead-beat " action. If the stimulus is removed, the reflex contraction stops at once — there is no after-discharge. Also, the stretch response to a tug or jerk on a muscle is restricted to the muscle, or even part of one muscle, which was stretched. In addition, the response latency is relatively short. Taken together, these observations suggest that the reflex arcs involved contain two neurons only — there are no internuncial neurons.

Stretch reflexes are most strongly developed in the extensor muscles, hence are of considerable practical importance to the organism in that they aid in resisting gravity. Thus, if one " sags at the knees," his extensor muscles are stretched and will contract reflexly. Even in men whose spinal cords have been cut, these reflexes eventually recover and many such patients have learned to walk on crutches and with legs made rigid by stretch reflexes.

The Extensor Thrust. The stretch reflex involves only the muscle stimulated, so that simultaneous stretch reflexes in all of the muscles of a limb must depend upon simultaneous stimulation of each individually. The **extensor thrust** is a reflex involving an entire limb. In the laboratory, the extensor thrust is readily shown in the spinal dog, preferably in the hind limbs, and consists of a brief thrust or extension of the entire limb following either pressure on the toe-pads or quick separation of the toes. The receptors most directly involved are the proprioceptors of the toe muscles, but pressure receptors in the skin are also of importance. In the intact organism, the extensor thrust is involved in walking and especially galloping. Here, when the foot strikes the ground, the extensor thrust comes into play, straightening the leg and driving the animal forward.

Reflex Coordination. In the discussion of muscular activity (Chap. 5), it was found necessary to introduce the concept of reciprocal innervation. This concept, you will recall, refers to the inhibition or relaxing of antagonistic muscles under certain circumstances. We may extend this basic idea somewhat to include reflex coordination of greater scope. Thus, in the spinal animal, both the flexion and extensor thrust reflexes cannot be aroused simultaneously in the same limb. One or the other reflex becomes dominant while the other is inhibited. If the stimulus is strong enough to arouse nociceptors, flexion will be dominant and the extensor muscles will lose in tone. Moreover, flexion reflexes do not occur in two limbs at the same time. As a matter of fact, a flexion reflex leads to what is termed a **crossed extensor reflex.** That is, if the left hind leg of the dog is flexed, the right hind leg displays an increased extensor tone. This increased tone arises fairly slowly and has a prolonged after-discharge. Under some circumstances of simultaneous stimulation on the two sides of the body, alternating contraction and relaxation in symmetrical muscles of the opposite limbs may be aroused and stepping motions result. This type of alternating thrusting and withdrawing can be demonstrated not only in the spinal dog but also in the newborn infant. Holding the infant erect and touching the feet to a flat surface will lead to stepping movements.

A caution needs to be interposed here, since it must not be thought that all influences from one side of the body to the other are of this alternating variety. Under some circumstances, simultaneous similar responses can be aroused on both sides as, for example, is seen when both hind limbs in the spinal dog exhibit strong extensor tone. Moreover, in the intact organism strict reciprocal innervation is not a necessary rule. This point will be discussed in more detail in Chapter 18.

Inhibition. Although we have used the term inhibition upon occasion, our discussion of reflex coordination requires its closer scrutiny. The basic observation is fairly simple. For example, in reciprocal innervation, if a flexor for a given joint contracts, the extensor actually loses tone. Some connections to motor neurons must be excitatory, therefore, but others must be inhibitory, that is, they actually interfere with excitation by other neurons. Inhibition of course, is not confined to the CNS. The chapter on the autonomic nervous system (Chap. 17) contains many examples of inhibition outside the CNS — the slowing of the heart by the vagus nerve, the stopping of peristalsis in the intestine by the sympathetic nervous system, and so on. However, our discussion here will be confined to inhibition as it relates to synapses.

Certain characteristics of inhibition in the CNS are rather well established, particularly with regard to the spinal cord. First, inhibition, just as excitation, exhibits occlusion or summation depending on the circumstances. Second, varying the strength of the stimulus and, hence, the size of the afferent volley will lead to variation in the amount of inhibition found. If, for example, we gradually increase the strength of stimulation of a nerve which leads to inhibition of a reflex which is being elicited by another nerve, the response is reduced as the inhibitory stimulation is increased. Third the same afferent nerve can excite one response and inhibit another at the same time. This is apparent from a consideration of reciprocal innervation. Fourth, inhibition may have considerable duration — as much as two seconds — which may be considered to be analogous to reflex after discharge and to imply long or reverberating circuits. Fifth, inhibition may have brief latency, as brief, in fact, as the latency for excitation. This implies that the connections for inhibition must be comparable in complexity, or simplicity, to those for excitation.

Although speculation has been rife, we simply do not know how a neuron can inhibit another or how the characteristics noted above can be explained. Just as in the case of synaptic transmission (Chap. 3), inhibition has been ascribed by some to a chemical process (humoral theory) and by others to an electrical phenomenon. The humoral theory is basically simpler and states that some neurons (or some endings of some neurons) secrete an inhibitory substance. Since it is well established that just such secretions occur at the peripheral endings of antonomic fibers in various bodily organs (Chap. 17), this idea has some plausibility. An electrical theory requires more elaboration since it is not easy to see how, if all nerve impulses are essentially alike, one neuron should excite across a synapse while still another should inhibit. This difficulty requires supplemental theorizing, usually of an anatomical nature. No theory is well established. The concept of inhibition rests on firm ground. The basis for it remains for future elucidation.

Long Spinal Reflexes. When the entire spinal cord is left intact, the animal can exhibit rather complex patterns of response, indicating a considerable degree of organization within the cord. Two carefully studied long (intersegmental) reflexes will be considered here. First is the familiar scratch reflex of the dog. If a spinal dog (or normal dog, for that matter) is stimulated by a vibration on the skin along the upper part of the chest, the hind leg of the same side will display scratching movements. At the same time, appropriate supporting responses are made in the other legs. It must be remembered that the response, including the rhythmical scratching movement, is organized entirely within the spinal cord. The second example of a long spinal reflex is the pattern of response which underlies walking in quadripeds. Harmonious rhythmic movements of the limbs are at least partly organized in the spinal cord, although in the intact animal considerable higher level control is also involved.

Visceral Spinal Reflexes. The spinal reflexes which we have so far considered involve the skeletal muscles. There are a number of spinal reflexes which involve the autonomic rather than the somatic nervous system. These include elimination and sexual activity which are mentioned in the next chapter. Visceral reflexes are under the influence of higher centers just as are the somatic reflexes. Other nervous activity can influence the knee jerk and it can affect elimination and sexual reflexes as well.

Spinal Man. Recent advances in medical technique have made it possible for persons with severed spinal cords **(paraplegia)** to survive and in many cases to lead active, productive lives. In such cases, if transection of the cord is complete, those portions of the cord below the cut are removed from the influence of higher centers and the activity of the effectors they serve is purely reflexive. Immediately after transection, reflex activity, as a result of the shock of injury, is absent. In a few weeks, flexor reflexes appear and finally extensor reflexes appear. Of course, sensation below the cut is permanently lost. Visceral reflexes may become active within a few days and the patient develops an "automatic bladder," that is, the eliminative reflexes empty the bladder when internal pressure has reached a given level. The possibility of using extensor reflexes to support the weight has been mentioned earlier in this chapter.

The severity of symptoms and the chances for recovery are, of course, markedly influenced by the level of transection. If the section is above the fourth cervical segment, breathing promptly stops because the nerves to the muscles of respiration leave the cord at this level. A common football injury is dislocation of the neck at about this level. Prompt artificial respiration will save the player if the cord has not been too severely damaged. Sections below this level will affect varying amounts of the body. In general, the lower the section the better the chances for survival.

Higher Level Reflexes

The reflexes of the medulla which are of most interest to us may be put under two general headings: (1) **vital reflexes,** and (2) **decerebrate rigidity.** We shall discuss the so-called vital reflexes first.

Breathing. The most dramatic of the vital reflexes is breathing. Although higher centers can influence the respiratory reflexes of the medulla, they cannot completely inhibit breathing activity. One can hold his breath for a time but he cannot commit suicide in this way. The reflex control of breathing is fairly complex and serves to illustrate how a series of interrelated mechanisms combine to give an integrated activity.

Normal, quiet breathing is based on active inspiration and passive expiration. As one breathes in, the diaphragm and rib muscles contract, forcing the viscera downward and bulging the abdominal wall, while the chest is elevated. When inspiration stops, the elasticity of the tissues serves to return them to their original position. The active inspiration is initiated by an inspiratory center in the medulla. This center is sensitive to the CO_2 and hydrogen ion content of the blood and responds directly to the condition of the blood. There is also an expiratory center which becomes active when more energetic breathing occurs, giving rise to active expiration. One may well inquire why inspiration stops at the end of a breath. If the inspiratory center is removed from all influences except the blood CO_2 it shows continuous discharge and inspiration is maintained. Therefore, an active process must intervene to inhibit the inspiratory center if normal, rhythmic breathing is to occur. This intervention comes from two sources. The first is from the distension of the lungs. When the lungs are distended, receptors therein are stimulated, leading to afferent impulses in the Xth cranial nerve (Vagus), which inhibit the inspiratory center. The second is from the action of a so-called pneumotaxic center. This center lies in the pons. It receives fibers from the inspiratory center and sends fibers to the expiratory center. When the inspiratory center is active, the pneumotaxic center is aroused; it, in turn, arouses the expiratory center and reduces the activity of the inspiratory center. This loop of connections is of importance largely after the vagus nerves have been cut. Such a loop, where the activity of a given center gives rise to processes which eventually influence the further activity of that center, is called feedback, a point which will be discussed more fully after we consider decerebrate rigidity.

Still another important mechanism contributes to breathing. This is based on the chemical receptors in the carotid artery — the large artery in the neck in which you can readily feel the pulses if you place your fingers lightly at the side of your windpipe. These chemical receptors respond to increased CO_2 or hydrogen ion concentration or decreased O_2 concentration. In normal quiet breathing these receptors exert, by way of their neural connections, only a slight tonic effect on the inspiratory center. In case the O_2 level of the blood falls and CO_2 content does not rise (at high altitude, for example), the carotid receptors provide some impetus to breathing.

The above outlines the basic mechanisms regulating breathing on the reflex level. Of course, higher centers can intervene and sometimes do to the detriment of normal health, as when the sighs of neurotic sadness overventilate the blood and lead to dizziness. Two important general concepts arise from our discussion. First, there exist central neurons which normally respond directly to the conditions of their environment, not only to impulses in other nerve fibers. Other examples will be discussed in relation to the antonomic nervous system and motivation. Second, an important way of maintaining equilibrium is by means of feedback loops, where the activity of some structure gives rise to events which ultimately help determine the further activity of that structure. The type of feedback which we have been considering is called negative because the ultimate effect is to reduce any deviation from equilibrium. For example, if blood CO_2 rises and respiration increases in depth and rate as the result of arousal of the respiratory centers, the increased respiration leads to a reduction in blood CO_2, thus ultimately reducing the stimulation of the centers and returning respiration to its resting level. On the other hand, a reduction in the vigor of respiration leads to a rise in CO_2 content of the blood which in turn arouses the respiratory centers.

Other Vital Reflexes. The " medullary animal " (one with the CNS severed between medulla and pons) displays several other rather complex reflex patterns involving not only the autonomic nervous system but, also, sometimes involving the simultaneous discharge of the somatic system as in the case of respiration. One such pattern is the maintenance of the blood pressure which depends upon reflex contraction of vascular musculature, largely aroused by stimulation of receptors in the carotid sinus. This is another example of a negative feedback relationship. More complicated patterns are sneezing, coughing, vomiting, and swallowing. These patterns are well-organized in the medullary animal.

Decerebrate rigidity is a pattern of intense, persistent extensor contraction which arises when the CNS is sectioned at the level of the midbrain. The highest structures necessary for this rigidity to develop are the **vestibular nuclei,** which receive impulses from the non-auditory inner ear (cf. Chap. 9), and from the **recticular formation.** The latter is a diffuse column of cells running through medulla, pons, and midbrain, as far as the diencephalon. Both the reticular formation and the vestibular nuclei exert an energizing influence on the lower motor neurons and lead to exaggeration of the stretch reflexes. Decerebrate rigidity arises, then, because positive feedback occurs in the stretch reflex arcs. This can be interrupted by cutting the muscle away from its attachment. The rigidity will disappear at once, because the stretch receptors will then no longer be aroused by the contraction of the muscle. In the normal animal this exaggerated positive feedback does not occur because there are inhibitory influences from higher centers.

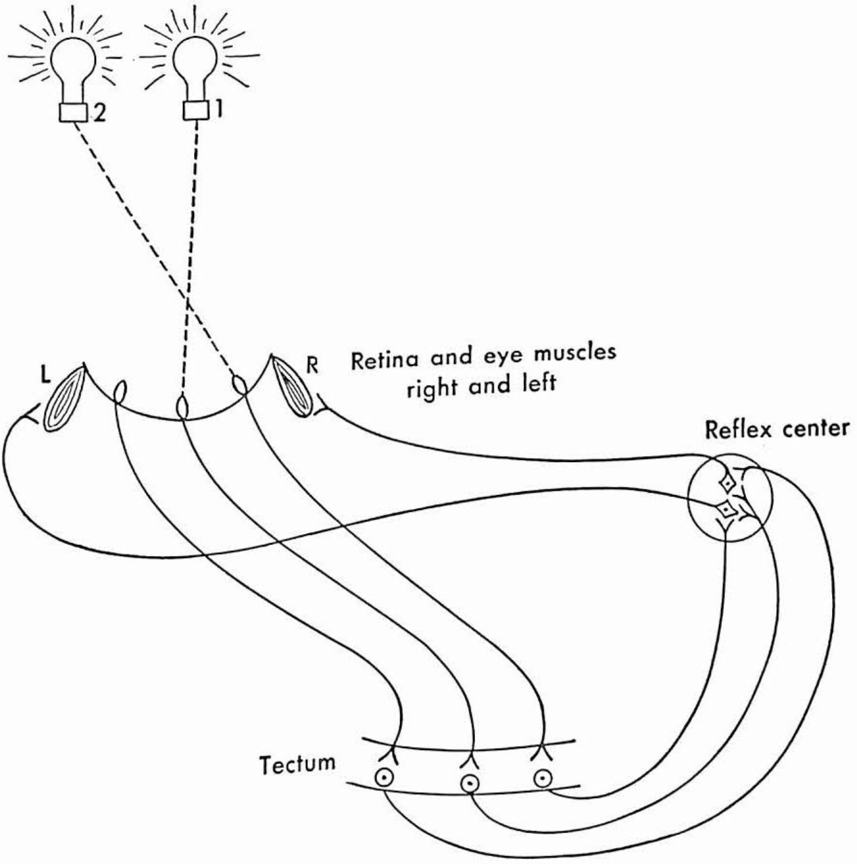


FIG. 16.5. Diagram of eye-aiming mechanism, one side only. Note how light No. 2 leads reflexly to aiming.

Aiming Responses of the Eyes. In the cat, the superior colliculi mediate an aiming response of the eyes which will serve as an introduction to a concept — the **servomechanism** — which has considerable currency in psychological theorizing. This term is a general one referring to feedback-controlled systems in general. First, let us consider the experimental results. If a cat has the structures above the midbrain removed, the eyes will automatically fixate a bright spot of light on a dark background. It is further found that if the superior colliculi (sometimes called the optic tectum) are removed, this aiming response is lost. Now it is known from anatomical research that the nervous connections from the retina preserve their spatial relationships in the outer layers of the tectum. Thus, each point of the retina is represented by a corresponding point in the tectal "map." Figure 16.5 diagrams this relationship. It can be seen that when the spot of light falls on the fovea (we consider only one eye for simplicity), the nerve impulses resulting will reach the tectum in the middle of the "map," point F. If the light falls to one side, the impulses arrive at some other point and reflexes are set into action which move the eye until proper aim results in the impulses reaching point F; that is, the eye is turned so that the spot of light falls on the fovea. Such a system is called a servomechanism in engineering and our physiological example is very similar in form to such mechanical devices as automatic pilots for boats and aircraft. Indeed, it would not be difficult to build a box on a pivot which would always turn its "face" toward a light. Since its introduction into psychology and physiology, the concept of the servomechanism has been applied to many nonreflex situations, such as aiming a gun, in which the activity of the organism is based on the relationship in space between the organism and some external target — bullseye, airplane, baseball — and in which the activity of the organism changes this relationship. You will observe that this situation involves feedback; in this case, through a loop of events actually going outside the organism. This type of activity will receive further consideration in subsequent chapters.

Higher Level Postural Reflexes. Some aspects of the maintenance of posture have been so thoroughly studied that it is possible to outline the reflex activity underlying them. Most of the work has been done on animals, particularly the cat, and the results do not hold in detail for man. We can, however, hazard the guess that in man there is considerably more encephalization than in the cat by observing the results from the study of monkeys. The reflexes may be present, but they are not as efficient and will not permit as precise adjustment. Some of the more important reflexes are outlined below.

If only the medulla is left connected to the spinal cord, the animal will be able to support itself although showing decerebrate rigidity. If now the head is turned (by the experimenter), lifted, or depressed, alterations in posture occur. These result from reflexes arising from receptors in the neck muscles and inner ear. When the head is turned to one side, extensor tone is increased on that side and decreased on the other. When the head is lifted the front legs are extended and the rear flexed. The reverse holds for depression of the head. These reflexes may be considered as useful parts of total bodily activities associated with the orientation of the head. Similar responses to head turning can be demonstrated in man.

If the midbrain is left intact, the animal exhibits "**righting reflexes.**" When placed on its side, the dog or cat will gradually, beginning with the head, assume an upright position. Primates possess the reflexes but do not achieve a standing position — greater encephalization has occurred. If a midbrain cat is dropped upside down, it flips over with amazing speed. This bit of coordinated acrobatics is accomplished by a sequence of reflexes. First, the head is turned by reflexes based on inner ear stimulation. Second, the turning of the head leads to stimulation of proprioceptors in the neck muscles which results in turning movements of the body. If the inner ears are destroyed, the animal drops on its back — unless the eyes are functional, in which case optic reflexes initiate the response. In fact, the eyes undoubtedly play the predominant role in the maintenance of posture in man, the inner ear being a poor guide. Nonvisual righting reflexes are not a good substitute for instruments when flying in clouds.

The midbrain animal also shows postural reflexes depending upon stimulation of pressure receptors in the skin. Since cat and primate reflexes differ somewhat, we shall describe the posture assumed by a monkey when placed on its side. Here the lower limbs are extended, the upper limbs flexed. Furthermore, there is a grasp reflex of the hand. If one places a weighted board on the upper surface of the animal, the postural reflex disappears. The grasp, however, is influenced by other righting reflexes and does not depend solely on the skin.

If the cerebral cortex is intact, two other general reflexes appear. These are called **hopping** and **placing** responses. Briefly, hopping is the alternate lifting and re-extension of the foot in the cat when it is held with one foot touching a surface and it is then moved sideways. Placing has several forms, but can be exemplified by holding a blindfolded cat horizontally and bringing the forelimbs against the edge of a table. The normal cat promptly places both feet on the table. These responses depend upon the cerebral cortex.

The postural reflexes may be considered to be important substrata for normal behavior. They provide some of the automaticity of detail which seems to be characteristic of motor performance and must, therefore, be taken into consideration when attempting to understand behavior. When we discuss, in Chapter 18, a few aspects of designing equipment for maximum efficiency, we shall see that the normal complement of reflexes must be taken into consideration. It would not do, for example, to have a sharp pain in the foot serve as a signal for extending the same leg!

Tropisms. Whereas a reflex is relatively limited in scope, a **tropism** involves total bodily orientation in relation to some external stimulus. Where righting reflexes leave off and tropisms begin may be a very fine point indeed. Perhaps a universally known example will serve to clarify the meaning of tropism. As is common knowledge, moths tend to approach a flame. For precision, let us say that moths tend to approach a light. This tropism depends upon rather rigid nervous connections with which the moth begins life. There are many other examples of tropisms, and words have been coined to designate the type of stimulus on which they depend — whether light, gravity, contact, temperature, and so on. Such tropisms are common among the insects, and some, such as the tendency of rats to follow walls, may be noted even in mammals. Our particular interest is more general, however, and centers on the analysis of tropisms in servomechanistic terms.

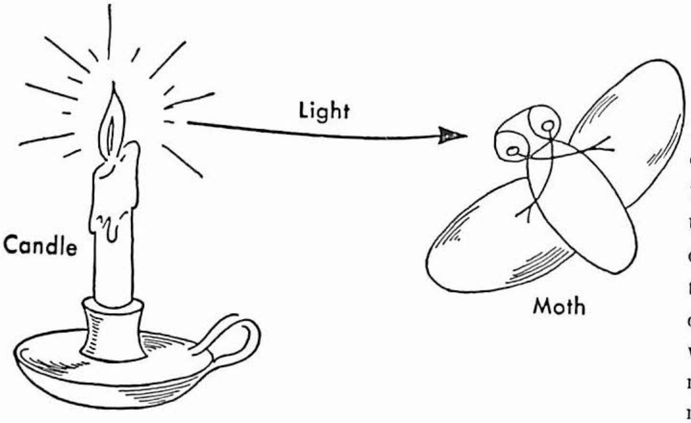


FIG. 16.6. Diagram of light tropism in the moth. Note how uneven illumination of the eyes can lead to increased strength of wing action in the wing opposite the more brightly illuminated eye.

Consider the moth. Let us postulate that the left eye, as diagrammed in Figure 16.6. is so connected to the nervous system that, when stimulated, it leads to stronger contraction of the right wing. The reverse is assumed to hold true for the right eye. If, now, one eye is more strongly stimulated than the other, the opposite wing beats more vigorously and the moth turns toward the source of stimulation.

The actual path will depend on this effect, plus the effect of the previous line of flight, and the moth describes a spiral. The similarity to the aiming response of the eyes is obvious and here again we have an example of a loop extending outside the organism, but this time involving the total animal.

Whether actual tropisms exist in man is problematical. Some avoidance behavior — leaving the region of a hot furnace, for example — seems similar to tropisms in other animals, (cf. Chap. 20), but such responses usually are not as automatic and invariable as classical tropisms. Nevertheless, it is possible to formalize some cases of very complex activity, such as aiming, in the same terms as tropism and, thus, to gain some advantage in the analysis of sources of error. We shall discuss this point in Chapter 18.

Suggested Readings

1. Lloyd, D. P. C., "Principles of Spinal Reflex Activity," J. F. Fulton, Ed., *A Textbook of Physiology*, 16th Ed. Philadelphia: Saunders, 1949.
2. Crozier, W. J., and Hoagland, Hudson, "The Study of Living Organisms," II, "Analysis of Conduct, Simple Tropisms," pp. 6–23 in Chapter I of Carl Murchison (Ed.), *Handbook of General Experimental Psychology*. Worcester, Mass.: Clark University Press, 1934.

17

Autonomic Functions

We come now to another important system for the integration of the functions of the body — the autonomic nervous system, or ANS. In the endocrine system, we found extensive means of chemical integration with little actual neural integration. In the ANS, we find another combination of chemical and neural integration but with the emphasis on nervous rather than on chemical functions.

In your study of the adrenal medulla and of the islet cells of the pancreas, you were promised more detailed descriptions of the functions of their hormones in relationship to the ANS. At least one of these glands, the adrenal medulla, is really a part of the ANS; and there is some evidence that the islet cells themselves also may be regarded as an effective part of this system.[1] It follows, then, that the ANS is intimately related to the endocrine system. Together they serve to regulate most of our internal functions and serve to maintain homeostasis.

There are several different points of view concerning the nature of the relationships between the endocrine system and the ANS. One holds that the endocrines are subservient to the ANS, whereas another holds that the endocrines control the ANS. Probably neither of these views is entirely correct. As a matter of fact, in single-cell organisms and even for a long time after cellular differentiation began to occur, most of the functions now served by both the endocrine and autonomic systems were taken care of by the chemical secretions of the cells themselves. Not until quite late in evolution do we find a true differentiation of secretory cells or of the nerve cells that make up the ANS. In the present state of our knowledge it seems pointless to consider which of the two systems is subservient to the other. Both are necessary in all warm-blooded vertebrate organisms for the maintenance of the internal environment, and it is difficult to consider the functions of one apart from the other. We saw, however, in our study of the endocrine glands that the posterior pituitary is under neural control from the hypothalamus. Moreover, there is some possibility that the anterior pituitary is influenced by secretion from the hypothalamus. Since the hypothalamus also serves as an integrator and controller of the autonomic system, one may think of it as a sort of " master controller " of homeostasis.

Terminology and Definitions. The ANS, or as it is sometimes called, the vegetative nervous system, is often thought of as a peripheral system of efferent fibers — that is, one that is outside the spinal cord and brain (CNS). Although any consideration of it must emphasize its peripheral aspects, it seems pointless to follow such an arbitrary classification. Most of the nuclei that control it are to be found in the brain stem and hypothalamus. Furthermore, we shall see that the cortex influences it too.

We may more appropriately think of three classes of peripheral nerve fibers, all of which are outposts of the CNS. One class is the sensory or afferent fibers; a second is the motor or efferent fibers that innervate striate muscles; and the third is the fibers of the ANS. The latter are motor; they innervate smooth muscles and glands; and — a quality which makes them unique — they synapse in ganglia between the spinal cord and the organs they innervate. Peripheral autonomic fibers, therefore, are of two sorts, **preganglionic** and **postganglionic.** Autonomic fibers also have another anatomical arrangement which is peculiar to them. Those that leave the spinal cord do so only at certain vertebrae instead of throughout the length of the cord.

Peripheral autonomic fibers can be divided into two classes with somewhat different characteristics. First, those fibers which leave the sacral region of the cord and those which leave the brain stem have long preganglionic processes, and the point of synapse is rather close to the muscle or gland which is innervated. Together they are known anatomically as the **cranio-sacral system.** Second, those that leave the central portion of the spinal cord (i.e., the thoracic and upper lumbar regions) typically have short preganglionic fibers which synapse with postganglionic neurons at a point not far from the cord. The anatomical name for this group is the **thoracico-lumbar system.**

The major ganglia of this latter system are interconnected by a number of extending fibers and form a chain on either side of the spinal cord known as the **sympathetic ganglionic chain.** These interconnecting fibers of the thoracico-lumbar ganglia are of great importance in understanding behavior. Such anatomical interconnection provides for mass action of the total system.

The thoracico-lumbar group of neurons attracted more attention in the early days of anatomy than did the cranio-sacral group, since it is interconnected anatomically to function more or less as a unit and since the secretion of epinephrine, produced by its own activation, facilitates its unitary action. It soon was given the name, the

sympathetic nervous system (SNS). By stimulation of this system, a number of organs were brought into function " in sympathy " with each other. Since the cranio-sacral system lies on either end of the spinal cord around the sympathetic system, it was natural for it to be called the **parasympathetic nervous system** (PNS). Today these remain the most popular terms.

The figure on the back-plate of this textbook will help you distinguish the two systems and should convince you that the vagus (pl. vagi; cranial nerve X) is the most important nerve in the parasympathetic system. The PNS represents a gradual specialization of those automatic activities of simple cells which are involved in the anabolic phase of metabolism. In other words, those activities in which the single-cell organism engages which are directed toward a conservation of energy or a restoration of the organism after exercise or tissue damage are, for the most part, the functions that are mediated by this cranio-sacral or parasympathetic system. It is obvious that these kinds of functions are essential to life. As organisms developed and became differentiated, this portion of the nervous system made its appearance quite early, but in simple organisms it is seldom called by a particular name.

The thoracico-lumbar system, on the other hand, may be thought of as having developed later in evolutionary history. Actually, we do not find it very clearly differentiated until we get to the higher organisms, especially the mammals, all of these being warm-blooded. In general, this group of nerves may be thought of as having functions opposed to those of the cranio-sacral group. They are, then, involved with the catabolic phase of metabolism. Another way to look at it is that they are concerned with preparing the organism for emergency reaction. They are not essential to life in a normal, well-controlled, environment if the organism is not subjected to unusual stimulation. In fact, there are many examples in scientific literature in which animals have lived for long periods of time under laboratory conditions in the absence of postganglionic thoracico-lumbar motor nerves, which are easily destroyed by removing the ganglionic chain and other major ganglia shown in the back-plate figure.

Together, these two groups of nerves mediate most of the functions of the visceral organs of vertebrate animals, including man. The two systems are in continuous activity. All organs which they both innervate usually are maintained in a state of balance or equilibrium. With a change in stimulation, the first reactions are usually among the thoracico-lumbar group. The nerves of this group, which you have seen to be hooked up together anatomically, respond more often than not as a unit. Furthermore, they respond to almost any kind of stimulation, but are particularly sensitive to stimuli we refer to as " noxious ". Since these noxious stimuli are believed by some authors to constitute the bases for our social motives, desires, and wishes, it is not unexpected that these nerves are brought into play by desires or wishes. We shall see later, when we come to the study of emotional behavior, that it is primarily this group of nerves which constitutes the basis for our understanding of emotional excitement.

Chemical Characteristics of the ANS. All ANS nerve fibers liberate chemical substances at their terminal endings and the organs they stimulate are reactive to these chemicals, whether from the nerve fiber or from the blood stream. Nerve fibers of the cranio-sacral system are reactive to acetylcholine and they liberate the same or a similar chemical. Thus, such a chemical substance in the blood will serve to mimic parasympathetic activity. Furthermore, there is evidence that at least one of the major nerves of this group (the vagus) stimulates the liberation of insulin from the islet cells of the pancreas which in turn, facilitates in some manner the responses of organs under parasympathetic innervation.[2] One investigator, as a result of this evidence, suggests that we speak of the " vago-insulin system "; and he would contrast it with a " sympathetico-adrenal system." There is some justification for this point of view as we shall see.

The fibers of the sympathetic system also liberate chemical substances at their terminal endings. Although the preganglionic fibers of this system are reactive to and liberate acetylcholine, the postganglionic fibers, for the most part, liberate another chemical which is similar to the epinephrine secreted by the adrenal medulla. This chemical substance is known as sympathin, and it is believed that two forms of it exist: (E), excitatory, and (I), inhibitory. They are referred to as **sympathin E** and **sympathin I.** It is known also that almost all of the organs stimulated by sympathetic fibers are reactive to epinephrine or norepinephrine in the bloodstream. You may see, then, that we have in this group of nerves and end organs the possibility for unified integrated action in terms of both anatomical and chemical characteristics and that there is good evidence for the use of the term " sympathetico-adrenal system."

✓**Functions of the SNS.** In a description of this branch of the ANS, it will be of value if you think of a particular pattern of behavior, fear. How do you feel when you are afraid? Your mouth goes dry, your pupils dilate, you feel your heart pounding and racing, your hands and feet become cold and they sweat profusely. Your digestion stops, or at least is interfered with and you may feel a lump or " butterflies " in the stomach. Many other things happen and Table 17-1 presents a list of most of the functions affected. Fear is the best example we can give you of the effects of these sympathetico-adrenal functions of our body. Practically all, but not quite all, of these functions also can be elicited by the injection of whole adrenalin into the bloodstream; but one major difference is that under adrenalin there is little increase in perspiration, whereas in fear the sweat glands secrete because they are directly innervated by sympathetic nerve fibers. The difference is explained by the fact that most of the fibers that innervate the sweat glands are **cholinergic** (i.e., producing acetylcholine-like effects) and most sweat glands themselves are insensitive to **adrenergic** substances (i.e., a substance with adrenalin-like effects).

Table 17-1. Some Prominent Facilitative and Inhibitory Autonomic Functions

Structure	*PNS Effect*	*Function*	*SNS Effect*
Eyes Iris	+	Constriction	−
Lens	+	Accommodation	−
Lacrymal glands	+	Tears	−(?)
Nasal mucosa	+	Secretion, dilation	−
Salivary glands	+	Salivation	−(?)
Gastro-intestinal tract	+	Peristalsis	−
Stomach glands	+	HCl, pepsin, and mucus	0
Pancreas (islet cells)	+	Insulin	0
Heart (rate)	−	Acceleration	+
Lungs (bronchia)	−	Dilation	+
Adrenal medulla	0	Adrenaline	+
Peripheral blood vessels	?	Vasoconstriction	+
Sweat glands	0	Sweating	+
Pilomotor cells	0	Piloerection	+
Internal sphincters Bladder Intestine	−	Contraction	+
Bladder wall Lower bowel	+	Contraction	−
Genitalia	+	Erection	−

Note: In the table (+) indicates a facilitative effect and (−) an inhibitory effect. Note that the upper portion of the table emphasizes facilitative effects of the cranial parasympathetics, that the bottom portion separates the sacral parasympathetic effects, and that the central portion emphasizes sympathetic facilitative effects.

Table 17-1 and the back-plate figure, studied together, will give a better understanding of automatic function than could pages of reading. We shall emphasize certain characteristics, however, and begin by completing the autonomic picture of fear. Your mouth becomes dry because what little saliva is produced is thick or viscous. Your pupils dilate, not only because of inhibition of the circular smooth muscles of the iris, but due to active contraction of another group of radial muscles under control of the SNS. Your heart not only increases its rate but its output per beat and, with the constriction of peripheral blood vessels in most portions of the body, there is an increase in blood pressure and some of the blood is shifted to your muscles. The peripheral vasoconstriction, of course, is what makes you pale and makes your skin cold, and you feel this most in the extremities of the body. The increased sweating is confined largely to the palms of the hands, the soles of the feet, the arm pits, the groin, and in some people the forehead and upper lip. These are the so-called " emotional sweating areas "; and they differ from the " heat-regulatory sweating areas," which respond predominantly to high bodily or environmental temperatures.[3]

In these patterns of sweating we have one definite indication that the SNS does not always act as a unit. Another indication is in the **pilomotor response.** Although " goose-flesh," as you probably call it, with erection of the hairs (piloerection) may occur during fear, it is much more prominent when we are cold. That the SNS does not always respond as a unit, even though we emphasize its mass-action tendency, is of significance in the study of emotional behavior. We shall refer to it again in Chapter 21.

Other definite effects occur in fear. Epinephrine is liberated in marked quantities and not only induces a prompt liberation of blood sugar from the liver but circulates in the blood stream and reinforces most SNS effects. The passages in your lungs (**bronchia**) dilate and the membranous linings of your nasal passages (**nasal mucosa**) contract and reduce their secretion, so you can breathe more freely — a fact that is easily noted if you have a cold or are asthmatic. Also, you breathe more rapidly, your eyes become dry and better adapted for far-vision, and your digestive processes are arrested; that is, muscular contraction (**peristalsis**) in the entire gastro-intestinal tract is inhibited. Note, also, that contraction of the bladder wall and bowel is inhibited and the internal **sphincters** or rings of smooth muscles at the outlets of the intestine and bladder are contracted. These conditions set the stage for retention of urine and fecal matter, not for evacuation. We shall consider later how one may explain the defecation and urination that sometimes are reported during fear.

It will be apparent to you that most of these SNS effects prepare your body for vigorous action, prepare you to escape from the source of your fear, or to give battle if necessary. Therefore, many authors consider that the SNS is primarily involved in preparing organisms to respond in emergencies. This concept is acceptable if one is careful to use the term " emergency " in a very broad sense. For example, other stimuli that throw the SNS into more or less mass action are extremes in temperature, hunger, exercise, pain, or bodily injury. It should be emphasized, however, that the SNS is in a continuous state of low-level activity opposed by, but in balance with, the PNS; and its activity is increased by almost any change in stimulation. Ready examples of this latter statement are found in measurements of blood pressure, peripheral blood volume, or galvanic skin reflexes **(GSR)** which indicate minute increases in sweat secretion.

Functions of the PNS. In preceding paragraphs we have seen that the SNS has extensive anatomical interconnections and that, by its stimulation of the adrenal medulla and the resultant secretion of epinephrine, its effects are facilitated chemically. There is good reason, then, to speak of a sympathetico-adrenal system. There is less reason, however, to speak of a vago-insulin system, or even of a parasympathetic nervous system. It makes more sense to consider parasympathetic effects as brought on by individual reflexes of a group of nerves which have something in common anatomically and chemically and which, under certain conditions, seem to respond more or less at the same time. One of these conditions seems to be SNS activity itself, and it is possible that such integration as the PNS then shows is under the control of the hypothalamus. Evidence is accumulating, however, that the vagus-insulin relationship is comparable to the sympathetic-adrenalin relationship. It has been shown, for example, that insulin secretion is increased under vagus activity and it, in turn, seems to facilitate in some manner the effects of vagal stimulation and even the effects of other cranial nerves. Perhaps there is a chemical integrating factor represented here; but much research needs to be done before we can speak of its relationships as definitely as we can of those between the SNS and epinephrine.

There is no single example in ordinary behavior patterns that will serve to illustrate general parasympathetic activity, but the patterns of presleep and sleep approach it. Table 17-1 shows the pattern. The pupils of the eyes are constricted, there is continuous secretion from the tear glands and the nasal mucosa (witness the eye-wiping and nose-blowing upon awakening), and frequent swallowing indicates the continuous secretion of saliva. Peristalsis is continuous (and may even result in rumbling noises), the heart and respiration rates are slow, the blood-pressure low, the bronchia of the lungs constrict and may make breathing more difficult, and hydrochloric acid is secreted by the stomach as any sufferer from stomach ulcers can attest. Before bladder and bowel training, defecation and urination are more frequent than when awake, and in those children with whom difficulty is experienced in training, most of the difficulty occurs during sleep. Furthermore, spontaneous erections during sleep are common among healthy males, and similar tissue changes probably occur also in the female. The pattern, for the most part, is seen to be one of conservation of energy. How much of it is attributable to increased parasympathetic stimulation and how much is due to decreased sympathetic stimulation is a question that will not soon be answered. At least it demonstrates a pattern of relative parasympathetic dominance and it may help you to remember the major parasympathetic effects.

A number of examples illustrate that local reflexes are responsible for many parasympathetic effects. We cite a few of them: food-taking produces increased salivation, peristalsis, and HCL secretion; a deep inspiration causes slowing of the heart; foreign matter in the eyes brings tears; a full bladder or bowel elicits contraction of the muscular walls and inhibition of the sphincters, with resultant evacuation; and local tactual stimulation induces erection in the genitalia, a phenomenon brought about by vasodilation. Some such separate reflexes are also to be found among the sympathetic nerves, of course, but there they are exceptional; while for the parasympathetic nerves they seem to be the rule. When abnormalities exist in the PNS, then, we may find them only in particular parts of the body. ✓

Individual Differences in Autonomic Function. It will be apparent that, in so far as either branch of the autonomic nervous system responds as a unit, and in so far as these branches are not exactly balanced in their antagonistic activity, it will be possible for individual differences to appear in total autonomic function. Although individual reflex differences undoubtedly exist, four main classes of differences are conceivable: excessively strong sympathetic action, or weak parasympathetic action, both of which would appear when measuring total autonomic function as a relative dominance of sympathetic function; likewise, excessive parasympathetic function, or weak sympathetic function, both of which would appear as apparent dominance of parasympathetic activity. At the moment we have no adequate techniques for independent measurement of the two systems. All that we can do is measure the functions that are innervated by both systems and arrive at some estimate of the relative balance of the two.

Since the turn of the century a number of authors have spoken of individual differences in autonomic functions. Some employ the term **vagotonia** for apparent parasympathetic dominance, because of the major influence of the vagus nerve in this system; while they speak of **sympatheticotonia** to indicate apparent sympathetic dominance. The terms are not very good ones, however, for they disguise relative differences in balance and they emphasize extreme types that seldom exist. These facts account in part for the lack of success of early investigators in their attempts to measure vagotonia and sympatheticotonia. Interest in the possibility of finding and measuring differences in ANS function has persisted, however, for at least two reasons that concern us: (1) their possible relationships to personality and (2) their possible relationships to mental and psychosomatic disorders. Considerable research is being conducted in this area today.

It is possible to measure individual autonomic differences in two ways: (1) relative balance in a resting state and (2) patterns of reactivity. The attempts to measure relative autonomic balance in the resting state have usually employed numbers of subjects and many autonomic functions, and then have attempted, by statistical methods, to find some combined scores to represent ANS function. Since most of the autonomic functions that have been measured are innervated by both the SNS and PNS, it is to be expected that only one autonomic score is to be found and that measurements of it for a particular individual will show only his position in relation to the rest of the individuals on whom the study was based. It will show how much he deviates from the mean score for the group and the direction of the deviation. If we consider the mean score to represent autonomic balance in a population, then a deviation in one direction might be called " relative apparent sympathetic dominance " and a deviation in the other direction might be called " relative apparent parasympathetic dominance." This, then, is a statistical concept of autonomic balance, as contrasted with the physiological concept of autonomic balance, i.e., homeostasis, or physiological equilibrium.

In one statistical study in which autonomic scores were obtained, the distribution of scores for a group of 84 school-age children was found to be approximately **normal.** (Cf. Fig. 17.1) Thus, about the same number of children showed sympathetic dominance as parasympathetic dominance; but extreme children in the former group were found to be easily excited to emotional behavior while extremes at the parasympathetic end of the distribution were more calm. Similar scores have been obtained for aviation cadets[5] and there, too, the distribution was approximately normal. As compared with this standard group other groups of adults appear to be different in ANS scores. For example, psychotics,[6] patients with tuberculosis,[7] individuals with psychoneurosis of the anxiety type, those with asthma, and those who were returned during the last war with the diagnosis of "operational fatigue," [5] all have been found to demonstrate a relative dominance of SNS activity. Whether this is a causal factor or a result of their disorders is not certain, but there is evidence that both interpretations are valid. In other words, it would seem that relative SNS dominance, whether inherited or acquired, will predispose a person toward emotional excitability and even mental abnormality or physical disorder; and, likewise, those conditions will facilitate relative SNS dominance. We shall discuss some of these problems in Chapters 22 and 24.

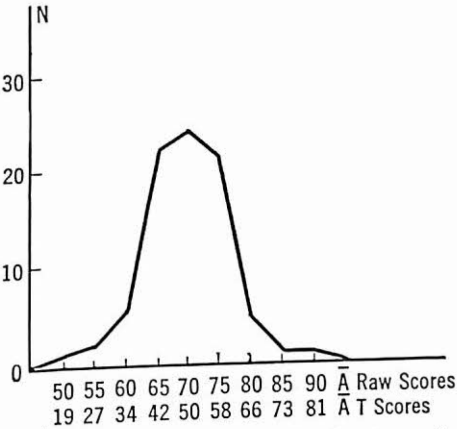


FIG. 17.1. Frequency distribution of mean estimates of autonomic balance ($\bar{A}$) for 87 children 6 to 12 years of age.

Patterns of autonomic balance do not tell the whole story, however, and must be supplemented by measures of ANS reactivity. For example, some persons who show more or less normal autonomic balance scores are found to be markedly deficient in SNS reactivity, while others demonstrate hyper-reactivity to stressful stimuli. Some show their greatest reaction in heart rate, others in blood pressure, and still others in sweating.[1] The significance of such individual differences is not yet understood but is the object of much research. Recent studies indicate that they are important in deciding on the type of treatment in insanity,[8] in psychoneurosis,[9] and in cancer; [10] and their probable relationship to emotional behavior will be discussed in Chapter 21. Figures 17.2, 17.3, and 17.4 will give you some understanding of the methods used to study autonomic activity in man. We shall give further attention later to the significance of such research.

Parasympathetic Overcompensation. One of the characteristics of the ANS which is often neglected is its tendency to demonstrate a phenomenon that might be called "overcompensation." The SNS is the system that seems to go into action first following any intense stimulation and the PNS apparently serves to counteract, or to com-

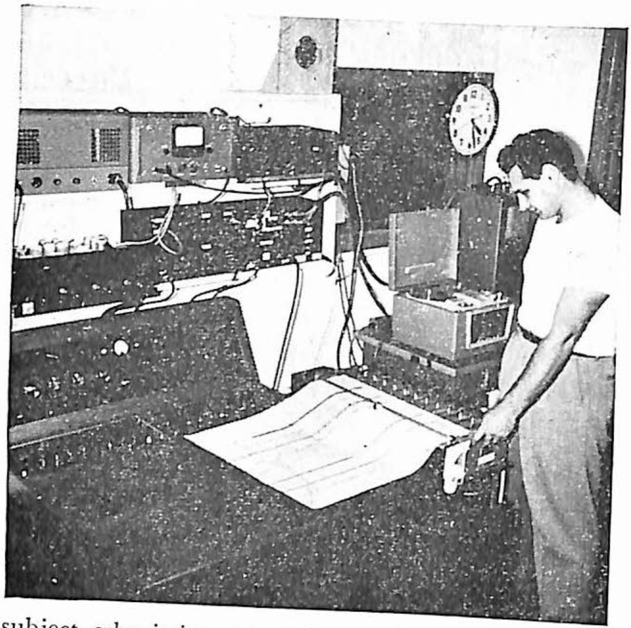


FIG. 17·2. The recording room of a laboratory at the Department of Psychology, University of California at Los Angeles, designed to investigate autonomic response patterns. The twelve pen-motors are activated by eleven Offner and one special amplifiers. At the far right is a Gilson automatic blood pressure recorder. Beside it is a tape recorder which, with the intercommunication speaker above, provides information on verbal responses of the subject, who is in a room beyond the window (see Fig. 17·3). (Photograph by Dr. William Calvert.)

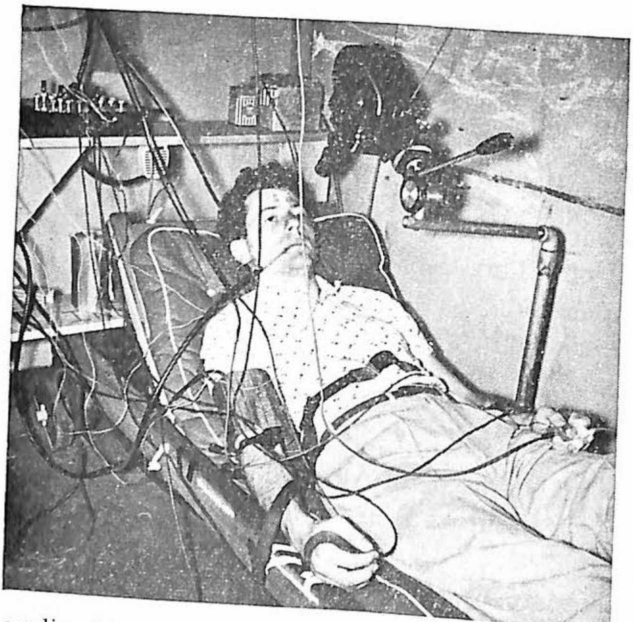


FIG. 17·3. A subject in an electrically shielded room adjoining that shown in Figure 17·2. Forehead electrodes are for an electromyogram. thermistors are taped to the right cheek and middle left finger to detect skin temperature, and the tube in the mouth withdraws saliva. Also visible are the blood pressure cuff and microphone on the right arm; the strain gauge which detects respiration; a cup on the left forefinger for recording blood volume changes; the straps which hold electrodes to the palms of the hands for skin resistance measurements; on the left arm one of two electrodes for detecting heart potentials; and the camera which records pupillary diameter. (Photograph by Dr. William Calvert.)

pensate for, this increased sympathetic activity. In some persons, presumably those who possess strongly reactive parasympathetic systems in part or totally, the activity of this system not only completely compensates for the increased sympathetic activity but, upon occasion, may overcompensate. This phenomenon is well illustrated by the following example. It will be referred to as **parasympathetic overcompensation.**

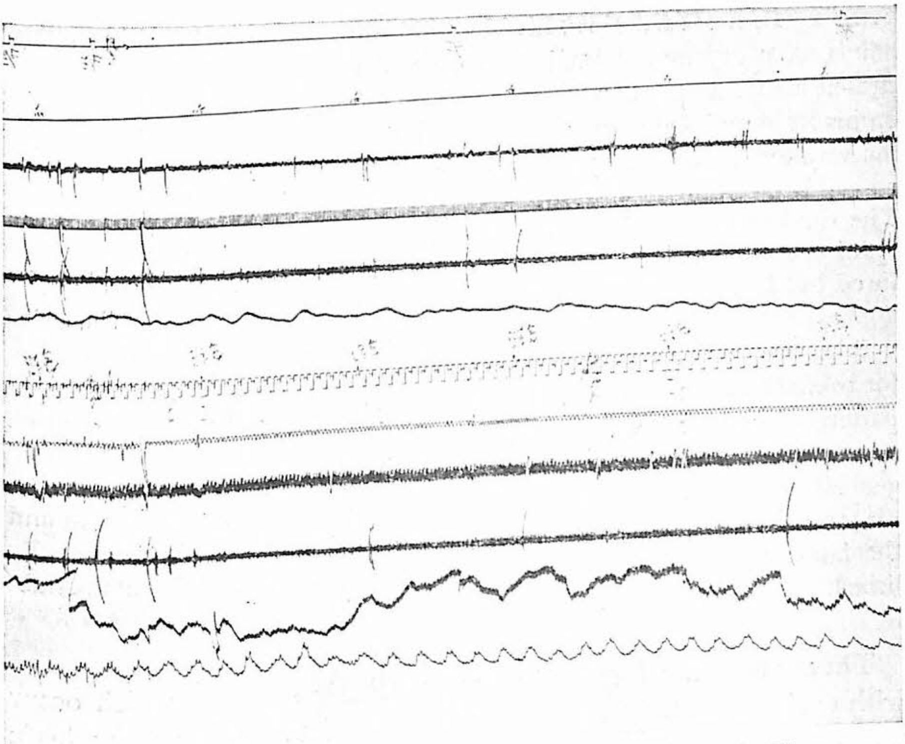


FIG. 17.4. Sample of records obtained in the laboratory shown in Figures 17.2 and 17.3. Chart speed is 3 inch per min. The twelve channels of data represent:

(1) Signal line (signals indicate time of blood pressure recording, and incidental information, such as onset and termination of stimulation);
(2) Palmar skin resistance, measured in units of 1000 ohms;
(3) Electromyogram, forearm flexor muscle potentials;
(4) Electrocardiogram, the electrical potentials of the heart beat;
(5) Electromyogram, forearm extensor muscle potentials;
(6) Leg plethysmogram, indicates blood volume changes in leg;
(7) Skin temperature – finger, axilla, and face (intermittent samples);
(8) Respiration, as measured by an electrical strain gauge;
(9) Cardiotachometer, a measure of irregularity in the heart beat;
(10) Ballistocardiogram, a rough measure of the stroke volume of the heart;
(11) Finger plethysmogram, indicates blood volume changes in finger;
(12) Stomach contractions, obtained by remotely detecting the movement of a small magnet which the subject has swallowed.

John was a slender, ten-year old boy, who had demonstrated over a three-year period one of the lowest scores on the scale of autonomic balance to be found in a group of eighty-four children. He showed all the signs of chronic hyperactivity of the sympathetic nervous system: a fast heart, a high blood pressure, a dry mouth, perspiring hands, etc. The experimenters who were making annual measurements on this group of children had come to expect John to show this kind of physiological pattern at each measurement, and so were greatly surprised when, at the occasion of one annual examination, he showed just the opposite kind of pattern. His heart was slow, his blood pressure was low, the palms of his hands were dry and showed high electrical resistance, and he demonstrated a copious salivary flow. It was noticed that John had a bandage on his head and the discovery was made that he had had an accident on the way to the laboratory. One of the doors of an automobile in which he had been riding had not been securely fastened and he had fallen out. The car had been going slowly at the time of the accident and, in the opinion of the driver and the physician, John had not been seriously injured *but he had demonstrated intense fright.* The accident had occurred approximately one hour before the measurements described above.

John was returned to the laboratory in late afternoon of the same day for re-examination, at which time it was discovered that his characteristic pattern of apparent sympathetic dominance had been re-established. Presumably, his intense fright in the morning had been followed by rather general overcompensation on the part of the parasympathetic system and his measurements thus showed a marked parasympathetic dominance. By the late afternoon his typical physiological pattern had been re-established.

There are many forms of parasympathetic function which occur with excitement, but excitement is presumed to involve sympathetic activity. For example, fainting typically occurs in some people when anticipating the experience of a needle stuck into the arm, or when witnessing an operation; but fainting involves decreased blood pressure. Asthmatic attacks typically occur in some individuals following intense emotional excitement in which the sympathetic system is certainly dominant, yet the acute asthmatic attack is characterized by congestion of the bronchia, a parasympathetic response, and is relieved by adrenalin. Another case in point is the increased peristalsis and secretion of hydrochloric acid which follows emotional stimulation in some people, a response pattern which is often regarded as one of the major factors in the development of stomach ulcers, and one which is a parasympathetic function.

All of these seemingly anomalous forms of reaction become explainable if we interpret them as sequelae of emotional crises, rather than as a part of the original emotional pattern. As a manifestation of parasympathetic overcompensation in individuals with differentially organized autonomic nervous systems, they no longer appear as chance reactions. Perhaps patterns of defecation and urination with emotional behavior may also be construed as examples of temporary overcompensation.

The Conditioned Response and Autonomic Functions. You probably have learned about the conditioned response in your study of introductory psychology and know that almost any stimulus can come to elicit almost any bodily response, after conditioning. The principles of conditioning were first described in terms of an ANS response, i.e., salivation; and many ANS responses have now been conditioned in many experiments: respiration changes, the galvanic skin response, changes in heart rate, pilo-erection, pupillary constriction, and so on. Some experimenters have demonstrated that pupillary constriction and vasoconstriction may be brought under voluntary control in the laboratory through an involved process of conditioning, and much the same phenomenon occurs when we think of a lemon or a pickle and feel the saliva rush into our mouths.

But the implications of ANS conditioning often go unnoticed. Actually, these phenomena are with us in our day-to-day life and they account for many of the differences in our behavior. When a newly wed returns home after his typical hard day at the office, the sight of his bride quickens his pulse, alters his respiration, and releases a bit of epinephrine; but he usually does not get stomach pains. Years later, if his marriage has been an unhappy one, the sight of his wife still may give him the " shot " of adrenalin, but the rest of the pattern has changed; his stomach now may contribute an extra " shot " of hydrochloric acid to irritate his ulcer a bit. These phenomena are conditioned responses of autonomic functions; and they are related to emotion and to physical disorders. We shall have more to say of them later. For the present, remember that we differ in our autonomic responses to certain stimuli. Differential conditioning explains some of those differences; others presumably are inherited, or acquired before we are born.

Suggested Readings

1. White, James C., *The Autonomic Nervous System.* New York: Macmillan, 1952.
2. Mitchell, G. A. G., *The Anatomy of the Autonomic Nervous System.* Edinburgh: Livingstone, 1953.

18 *The Organization of Movement*

We discussed the reflexive and tropistic aspects of overt behavior in Chapter 16. As pointed out there, behavior consists of a good deal more than a chain of elementary reflexes. We shall now consider the more complex problems of movement, beginning with a discussion of the CNS structures involved, followed by a consideration of the origins of coordination — whether innate or learned.

When we considered reflexes, it was convenient to start with the spinal cord and proceed step by step to higher centers. This approach is justifiable because reflexes must be studied by means of analysis and the simplest cases represent the greatest degree of analysis. But such an approach will not do for our present topic. In mammals in general, and especially in man, encephalization has progressed to such an extent that the normal function of any CNS structure can be understood only when it is considered in its inter-relations with the cerebral cortex. The cerebellum, the corpus striatum, the thalamus — all these and more must be understood in their relationship to the cerebral cortex. Consequently, our first consideration will be the cerebral cortex.

THE CEREBRAL CORTEX

It has been known since 1870 that electrical stimulation of the cerebral cortex can lead to movement. Although it was thought that movement could be elicited only from stimulation of the so-called motor area, area 4 of the diagram in Figure 18.1, it is now known that such is not the case. Area 6 in our diagram (the **premotor area**), area 8, and others yield movement. Such results provide evidence that many, perhaps most, portions of the cerebral cortex give rise to outgoing, motor nerve fibers. Whence these come and where they go will claim our first attention.

Pyramidal and Extrapyramidal Tracts, or Systems. One often reads of the **pyramidal** and **extrapyramidal** motor systems. At one time they were thought to be quite distinct and different in organization, with the pyramidal system the sole mechanism of voluntary movement. Although derived from a fairly specific and limited definition — namely, those fibers found in the **pyramids** — the term "pyramidal" has accumulated a good many supplementary implications, many of them quite wrong. We shall adhere to the simple definition. The pyramids are simply the place at which the huge motor tracts, which we shall call the **pyramidal tracts,** cross from one side of the CNS to the other. The pyramids are distinctly visible on the ventral surface, as shown in Figure 18.2. The fibers in these tracts arise from many areas of the cerebral cortex, *not* solely from the motor area (area 4). Fibers in the pyramidal tracts end in motor nuclei in the brain stem and spinal cord. There is considerable evidence that most do not end on motor neurons, but rather upon intermediate neurons which in turn synapse with motor neurons. Although most fibers in the pyramidal tracts cross at the level of the pyramids, a small percent do not, although some of these cross later in the spinal cord.

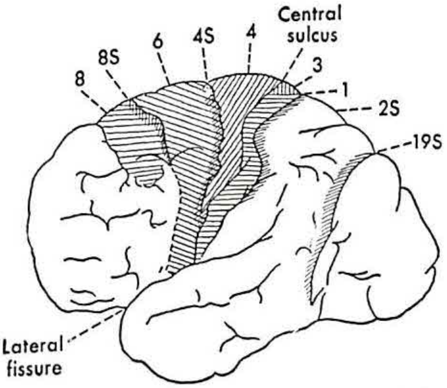


FIG. 18.1. Lateral surface of the left cerebral cortex showing the areas most directly concerned with movement.

All motor fibers outside the pyramidal tract are called, collectively, extrapyramidal. To assume that this term covers a homogeneous group of tracts would be utterly fallacious. We shall have occasion to discuss several "extrapyramidal" systems and when we have done so you will see quite clearly the justice of the above sentence.

The Motor Area (area 4). The classical motor area, located largely in the **pre-central gyrus,** has been very carefully and repeatedly mapped by means of electrical stimulation. It is possible, by very precise technique, to arouse a single muscle by cortical stimulation of this area. If we utilize this information to map the motor area in terms of the muscles aroused through stimulation, the map of Figure 18.3 results. Not shown is the fact that the effects of stimulation are largely crossed; that is, stimulation of the left side of the brain gives rise to movements on the right side of the body, and vice versa. It will be seen from the map that the representation is also "upside down," so that the head and arm areas are toward the ventral part of the cortex. It will also be seen that the size of the cortical area concerned with a given muscle is related to the fineness of movement possible in the muscle rather than to sheer bulk of muscle. Thus, the fingers and especially the thumbs are much more heavily represented than the legs or the trunk. In actual experimentation, a good deal of variability is found with repeated stimulation. Since even a sharply circumscribed stimulus probably influences a rather large number of intermingled cortical neurons, the precise effect of stimulation will depend upon a number of factors, among which are the effects of previous stimulation, the intensity of stimulation, and the time relationships between successive stimuli.

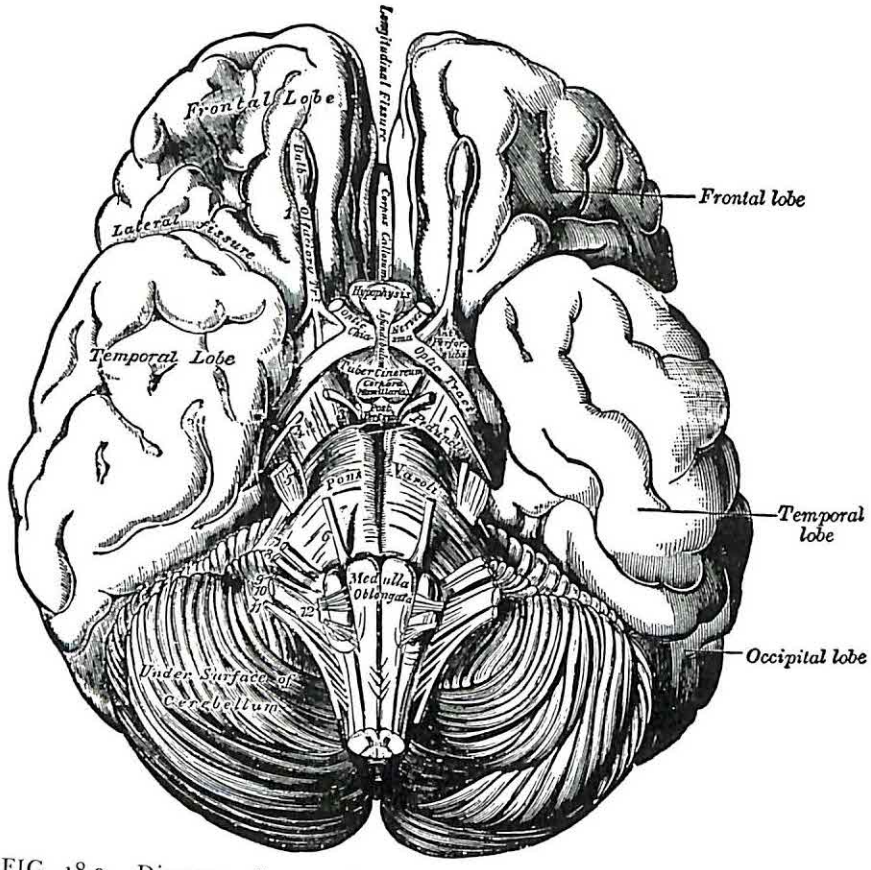


FIG. 18.2. Diagram of ventral surface of the cerebrum. The pyramids are labeled "medulla oblongata," of which they are a part. (From Kuntz.[1])

The removal of area 4 without other damage to the CNS results in a **flaccid paralysis** from which considerable recovery is possible, especially in the young organism. Since the pyramidal tract arises from many areas, not just area 4, it may be assumed that recovery is based upon the development of greater control by the neurons remaining after the operation. In any event, one must be careful to note that area 4, the classical " motor area," is by no means the sole point of egress from the cortex of nerve fibers concerned with voluntary movement.

The premotor area (area 6). It is more difficult to arouse movement by stimulation of the premotor area than by stimulation of the motor area. Their relative locations are shown in Figure 18.1. Despite the difference in excitability, it is not at all clear that the organization of the two areas is distinctly different. Removal of both areas, for example, leads to more severe symptoms than removal of area 4 alone. However, it is usually believed that area 6 is concerned with more widespread, postural types of response than those mediated by area 4. In any event, there seems little reason to doubt that area 6, as well as area 4, gives rise to fibers in the pyramidal tract and to fibers in the extrapyramidal system.

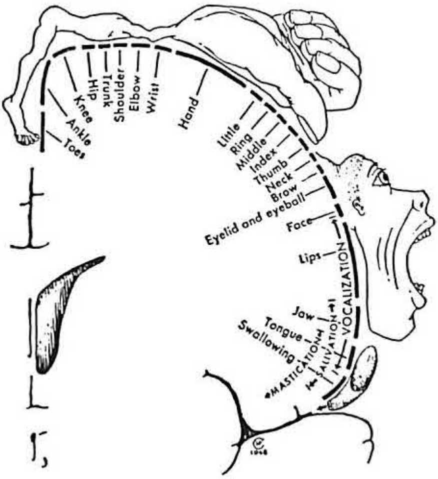


FIG. 18.3. Motor area of man as determined by electrical stimulation. The parts shown in the homunculus are those aroused by stimulation of the given cortical area. (From Penfield and Rasmussen.[2])

Other cortical motor areas. As we have indicated previously, motor fibers arise from many cortical areas other than 4 and 6. There is a heavy contribution from the somesthetic areas (1, 2, and 3), and from the so-called association areas. Thus, nearly the entire cerebral cortex is most intimately concerned with the control of movement. Understanding of this must wait for our discussion of the relationship of the cortex to other CNS structures.

Cortical suppressor areas. It has been found that stimulation of certain areas of the cortex — those labeled 2s, 4s, 8s, and 19s in Figure 18.1, as well as others not visible in a lateral view — will give rise to a reduction rather than an increase in activity in other cortical areas. At the same time, there may be a general relaxation of the skeletal musculature. This phenomenon has been termed **suppression.** Such suppression seems always to involve " loops " through subcortical structures and fuller discussion of these loops will be included when we discuss these other centers. However, it is proper to inquire at this point concerning the functional significance of suppression. In the first place, it is possible that the whole phenomenon may be an artifact of method, but we feel that the discovery of suppression from subcortical stimulation in certain centers lends plausibility to the cortical observations. Assuming the validity of suppression, it seems most likely that it is basically a homeostatic mechanism. If this is so, then suppression helps to smooth out the ups and downs of cortical activity, whether in small detail, as in controlled movement, or in general, as when severe pain arouses the suppressor areas and reduces cortical response. Needless to say, this idea is speculative and lacking in detail. But it does have the advantage of bringing cortical activity into line with other aspects of homeostatic activity in that balance is achieved through the interplay of opposing forces.[4]

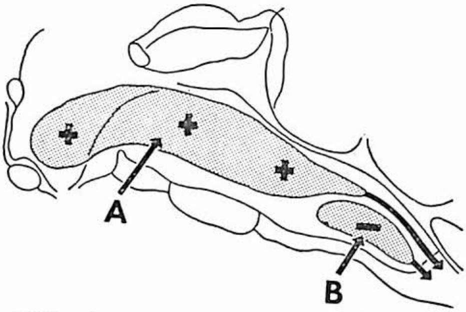


FIG. 18.4. Facilitatory and inhibitory areas of the cat brain stem projected on a longitudinal section of the brain stem. A is facilitatory, B inhibitory. (From Niemer and Magoun.[4])

SUBCORTICAL STRUCTURES

The Reticular Formation. The **reticular formation** is a rather diffuse column of scattered clumps of gray matter lying in the brain stem, as shown in longitudinal section in Figure 18.4. The lower portion, marked with a minus sign (—) in the figure, is a link in the system starting with area 4s which suppresses muscular activity. This is an example of a subcortical suppressor area. It works in close association with the cerebral cortex. The rest of the reticular formation, labeled with a plus sign (+) in the figure, is facilitatory and, also, has very important functions in relationship to wakefulness or alertness which will be discussed under those headings. In the realm of movement, we may note that the pyramidal tracts arouse the reticular formation and so this structure contributes to the energy of cortically initiated movement. Also, through the influence of the vestibular nuclei, it contributes to muscle tone. Lesions in the reticular formation will have consequences which depend upon the region — suppressor or facilitatory — affected.[6]

The Corpus Striatum. The corpus striatum, as indicated in Chapter 3, comprises the ventral and lateral portions of the cerebral hemispheres, although the cortex has so enlarged as to hide it completely from sight. Electrical stimulation of the corpus striatum in the resting animal leads to few observable effects. It is extremely likely that the striatum and the cerebral cortex act very much as a single unit, the role of the striatum being the smoothing out of cortically initiated activity. Observations of the effects of injuries to the striatum lend considerable weight to this view, since the results seem to depend upon the release of cortical activity from this smoothing influence. The neural connections between cortex and striatum provide an elegant example of a feedback loop — in this case, negative. The striatum receives fibers from the suppressor areas of the cortex and sends fibers to the thalamus (ventral lateral nucleus), which in turn sends fibers to areas 4 and 6 of the cortex (Cf. Fig. 18.5). Inter-

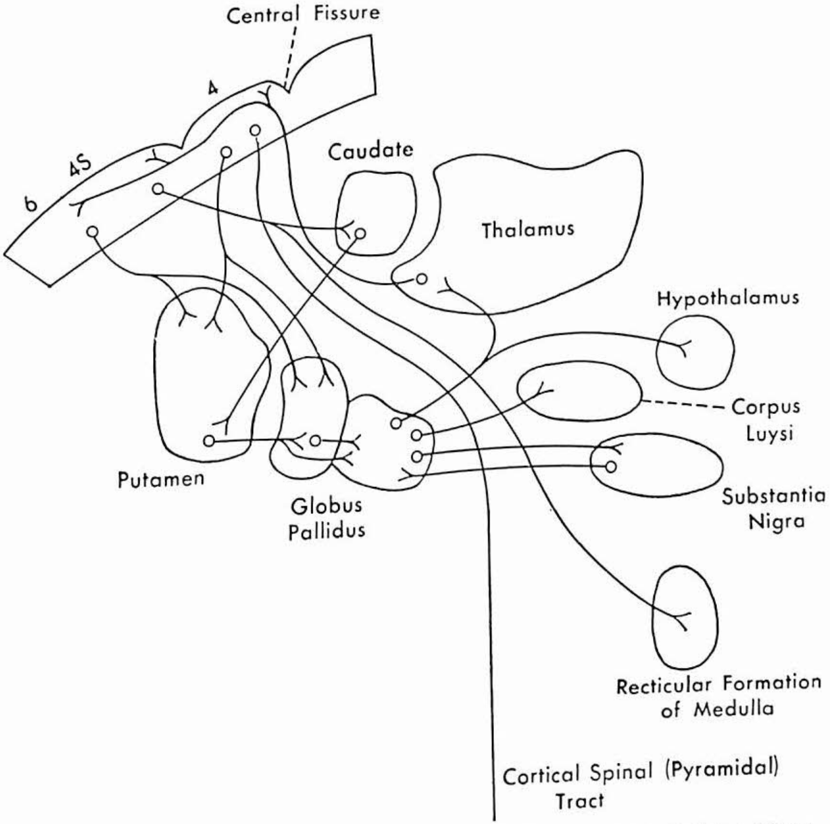


FIG. 18.5. Diagram of some of the subcortical motor connections. Note particularly the "recircuit" between cortex and corpus striatum. (From Ruch.[6])

rupting this loop gives rise to the release of the cerebral cortex with various symptoms of increased activity of the muscles, such as tremors and the circular movements of **choreoathetosis.**

The Cerebellum. The mammalian cerebellum acts almost entirely interdependently with the cerebral cortex, but it has no direct influence on the final common motor path. The cerebellum receives

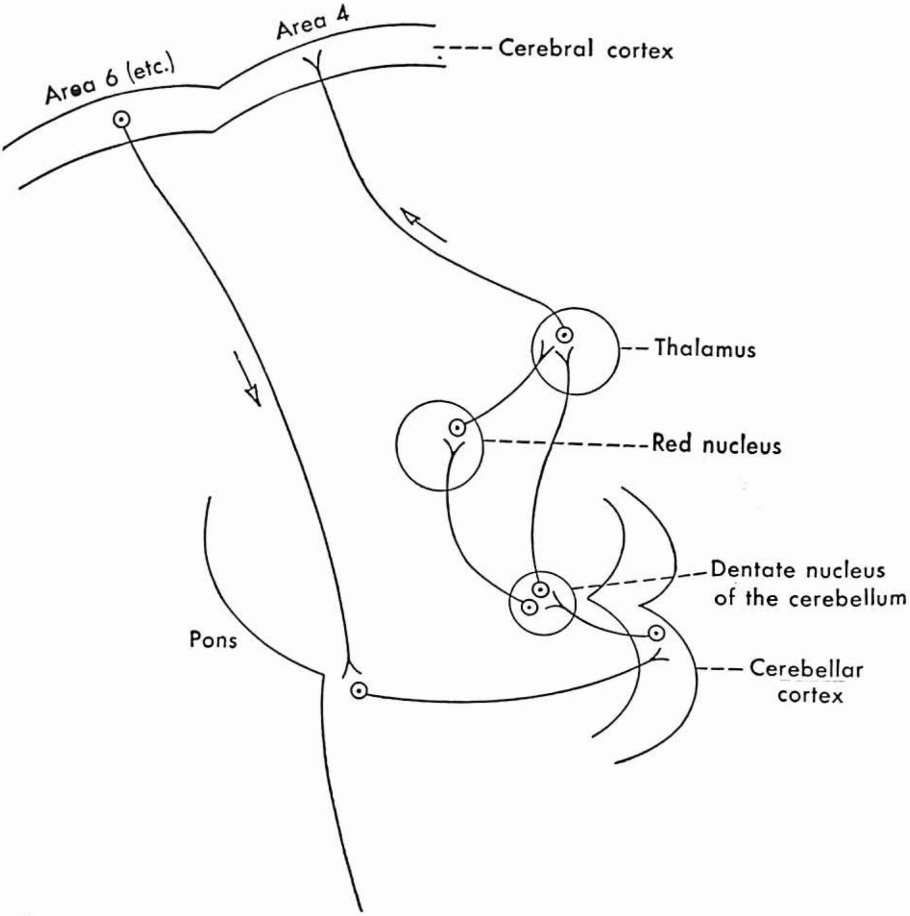


FIG. 18.6. Diagram of the motor relationship between cerebellum and cerebral cortex.

fibers from the various sensory systems and from the cerebral cortex. It sends fibers to the cerebral cortex and to various subcortical motor structures. The relationship to the cerebral cortex is illustrated in Figure 18.6 and is obviously of a feedback nature. The more primitive functions of the cerebellum have been mentioned in Chapter 9.

The motor function of the cerebellum is entirely one of strengthening and smoothing, since complete loss of the cerebellum weakens but does not destroy movement. Injury to the cerebellum will produce loss of muscle tone, tremor when movement is attempted, and errors in the rate, direction, and strength of movement on the same side of the body as the lesion. These symptoms are most readily interpreted as resulting from the release of the rest of the motor system from the smoothing and " metering " action of the cerebellum.

THE MOTOR SYSTEM AS A WHOLE

In this chapter and Chapter 16 the important neural components involved in movement have been analyzed. It now becomes necessary for us to attempt to recombine them in order to understand their contributions to the stream of movement. This task is not made any easier by the fact that all of the structures probably are functionally active all of the time so that the control of movement at a given moment depends upon a nice balance among a forbiddingly large number of variables. Ordinarily, the nicety of this balance is not appreciated until something goes awry and so we depend in considerable amount upon the clinic for our information. There is a very general principle, called Hughlings Jackson's Principle, which applies here. This principle states that the symptoms noticed when a given nerve center is destroyed are the result of the release of the remaining centers from the influence of the one removed. If, for example, injury to the corpus striatum leads to the strangely increased muscular activity seen in such a condition as choreoathetosis, this activity must result from the positive contribution of centers now removed from the restraining influence of the damaged structure. Such a view can have very practical as well as theoretical significance. For example, if the extreme **spasticity** (abnormally increased tone) of the muscles in such a condition as **paralysis agitans** is due to the positive activity of the remaining structures after damage to the corpus striatum, then removing the structure making this positive contribution should alleviate the symptoms — always assuming that some other worse symptom is not thereby produced. As a matter of fact, removal of area 6 (the premotor) of the cortex does reduce the severity of these symptoms; hence, the spasticity was due, at least in part, to the positive activity of a cerebral cortex released from the restraining influence of the feedback normally present from the striatum.

Our example leads us directly to an extremely important generalization concerning the way in which the motor system works. This is that the organizing centers in the normal animal are at all times supplied with feedback from other centers and from the muscles themselves. No motor center acts in isolation. Let us consider in more detail the particulars of this generalization.

Feedback from Other Centers. We have called attention to the importance of return neural circuits to the cerebral cortex from the corpus striatum and cerebellum. These operate in such a way as to smooth, meter, and strengthen movements initiated by cortical action. Without these neural loops, movement gets out of control and jerkiness, tremors, over- and under-shooting, and the like are clearly observable. It may be useful to put these effects under two headings. First, the feedback loops serve to help in the gradual and unjerky initiation of movement. They provide a physiological " fluid drive," which serves to restrain the effects of sudden changes in cortical motor patterns. Second, the loops serve to strengthen the effects of cortical patterns. They may provide a cascading linkage of neurons so that, instead of providing all of the innervation necessary for a given movement, the cortex can largely function by triggering a series of neural events which then provides the bulk of the neural discharge. These two aspects may seem contradictory, but they are not if one considers the timing involved. During the initiation of movement, the restraining influence is for a short time dominant while the strengthening effect grows larger later.

Feedback from the Proprioceptors. The fact that the receptors in muscles, tendons, and joints are of considerable importance has been pointed out in Chapter 9. We need only re-emphasize this point here. The motor centers are constantly informed of the events going on in the muscles and this information helps determine the future activity of these centers. Consider the experiment in which subjects learned to lift the hand from an electrode at the sound of a buzzer. When, after learning, the hand was turned over and placed on the electrode upside down, the subjects still lifted the hand at the buzzer signal, despite the fact that lifting the hand now meant moving it in exactly the opposite direction as far as the muscles themselves were concerned.[8] If feedback from the hand had not influenced the motor centers, this result could scarcely have occurred.

Feedback from Other Receptors. Our statement of the example in the paragraph above did not call attention to another aspect of the situation, namely, that the skin receptors were also involved. Perhaps you will remember that we pointed out in Chapter 16 that certain postural patterns were partly determined by the effects of pressure on the skin. These are examples of how touch can also serve to condition movement. But a very much more extensive generalization can also be made. Feedback loops can extend outside the organism, and a very large proportion of the material relating our concepts to the field of motor skill is based on the study of situations in which a goodly proportion of the feedback comes from loops outside the organism. Consider two children playing tag. Let us say that Johnny is " it " and Jimmy is trying to elude him. As Jimmy dodges, Johnny must immediately adjust his patterns of movement to keep " on target." As Johnny adjusts, his spatial relationship to Jimmy changes, so he must adjust some more. At any moment, what Johnny does next depends upon the visual cues — the visual feedbacks — which he gets from keeping his eye on Jimmy. In formal terms, we can call the relationship between Johnny and Jimmy a feedback loop. In other terms, we may say that Johnny's behavior depends upon the visual information which he is getting. No doubt you can think of other examples, not only for vision but for other senses, especially hearing.

The Role of Reflexes and Postural Patterns. Since we have discussed reflexes and posture quite extensively elsewhere, we shall not consider specific instances here. It is important, however, to understand how these inborn patterns are integrated with the rest of the motor system. In very general terms, we can point to two aspects of this integration. In the first place, the patterns of reflex action and postural adjustment obtaining at a given moment will affect the patterning of movement for the entire organism. Often this effect is merely a modification of some activity to accommodate the position of the body at a given moment; but sometimes, such as when a golfer loses his balance, the reflex action will take precedence and lead to quite disastrous results. In the second place, the reflex and postural mechanisms within the organism will influence the way in which a given act is carried out. For example, some acts seem more natural than others and in engineering it is very important to design equipment to take advantage of the natural movements of people. Also, the little details of integrated movement are taken care of reflexly. Patterns of walking, of maintaining balance, and so on, do not need constant higher level attention but are best left to the postural mechanisms.

The Control of Behavior. One may well ask why, if so many loops and structures are involved in movement, we should treat the control of movement separately at all. Is not the complete description of the control of movement also the description of the control of behavior? The answer to this question is, of course, yes. But there is profit in taking material in chewable bites, not only for the student but also for the scientist. We have endeavored, therefore, to emphasize those aspects of control which are concerned most directly with the execution of movement toward goals already set by prior processes in the organism. Our concern has been with how the patterning of movements is in general accomplished, rather than with the selection of the direction toward which the organism as a whole decides to go.

THE ORIGIN OF PHYSIOLOGICAL INTEGRATION

A problem which has intrigued psychologists and physiologists for some time is whether the basic patterns of integration in the CNS, and the relationships between these CNS patterns and the periphery, are laid down as a product of inherent growth or are developed through learning. In the past, both extreme positions have been upheld by one or another theorist. Where one insisted that even the most elementary reflexes are learned, another contended that even very complex social patterns are inherited. As so often happens, this debate has been largely silenced by empirical research, and we can assert with some assurance that the basic CNS and reflex patterns are growth-determined, while complex social processes are largely products of learning.

Growth within the CNS. Considerable effort has been expended by physiologists in studying the way in which neurons grow and, particularly, why they should grow in one way and not another during the growth of the CNS. When one realizes how many billions of neurons there are, he is probably somewhat astonished that connections are so seldom badly scrambled. A given nucleus of the thalamus sends fibers to a given cortical area; the large spinal conduction tracts somehow establish connections with the proper thalamic nuclei; and so on. Various lines of evidence now indicate that this is a matter of growth. The details are a matter of experimental embryology, far beyond our present concern. We may point to a few generalizations, however.

All neurons develop from the embryonic **neural fold.** This fold lies along the dorsal side of the embryo and soon forms a tube, the **neural tube.** This tube develops to become the CNS and from the cells within it come all of the neurons of the body. These embryonic neurons, called **neuroblasts,** send out the filaments which become axons and dendrites in mature nerve cells. Also, to some extent, they migrate short distances. The growing filaments tend to follow mechanical contacts — a membrane, for example, or some other surface. In addition to this tendency, there must be extremely subtle control by chemical interaction between the growing nerve filament and its environment. It is almost as though the flavor of the surroundings determines the specific ways of neuronal growth. Basic to both these control processes — contact and chemical — is the timing. The growing tissues are in constant flux, and the proper growth of neurons must depend upon a considerable nicety of timing. As a result of these processes at least the grosser anatomical relationships are formed, and there is evidence from the behavior of salamanders kept under an anesthetic during growth that the more particular connections on which reflex and other coordinated activity depends are also growth established.

Growth of Connections Between CNS and Periphery. Although a considerable amount of experimental work has been concerned with the innervation of the periphery, the bulk of it has been done on amphibians (salamanders and frogs) and the results should not be expected to transfer directly to mammals. The use of amphibians has been dictated by their rather high potentiality for regeneration. For example, a frog's eye may be excised, rotated through 180°, the severed ends of the optic nerve united, and then the optic nerve regenerates and the eye becomes functional![9] This experiment would fail in the mammal. It may be said, then, that the experimental zoologist uses the amphibian to study the processes of growth because of the great growth potential of even the adult, with the expectation that the results will be meaningful for other animals during their normal growth process. Of course, even in the amphibian, regeneration is not limitless and at some stage of development most structures attain sufficient specialization to limit regeneration severely.

The rotation of the eye in the frog provides a convenient example of one type of experimental result. This type we may call "receptor determined," and in this case the central connections are determined by the peripheral fibers. With the eye, this means that the fibers regenerating from the ganglion cells in the retina re-establish connections which were normal for them *before rotation*. The eye becomes functional, but the frog responds to stimulation in a manner which is now inappropriate, but which would have been appropriate to the old position of the eye. A similar result is found following section of the vestibular nerves. In the case of proprioception, the situation is very similar. The afferent fibers establish central connections which are appropriate to the muscle innervated. This is true even if a limb bud is transplanted to a new position and supplied with nerves from an unusual segment. Somehow, the muscle innervated imparts to the nerve fibers an individuality which determines their central synapses.[10]

Another type of result which we may call "afferent nerve induced" obtains for taste, even in mammals. Here, the afferent nerve fibers induce the formation of taste cells in the epithelium of the tongue. If a nerve serving taste buds is sectioned and permitted to regenerate, the associated taste receptor cells will degenerate and new cells will be formed when the regenerating nerve fibers reach the epithelium.[11]

When motor nerves are considered, the result is similar to that obtained with proprioceptive fibers — the muscle innervated determines the central connections of the afferent fibers. In the salamander, a limb bud may be rotated 180° and supplied with unusual spinal nerves. After regeneration, the limb moves as it would have moved in its normal position — even though by so doing the progress of the organism is seriously interfered with. In other words, no learning has been involved; the muscle has induced the nerves supplying it to make central connections appropriate to the muscle, not to the over-all purposes of the organism.

The Influence of Learning. If the result of research on amphibians can be generalized, we may say that at least the basic patterns of organization of the CNS are genetically determined. One must proceed with caution, however. Experiments by persons wearing eyeglasses which invert the visual field show that it is possible to learn new spatial relationships in vision which are reversed from the normal. Clinical procedures leading unusual nerves to paralysed muscles have been very successful, and the patient develops good control. It is even possible, by proper harnesses, to give an amputee good control over an artificial arm, despite the fact that the muscular contractions involved are very remote indeed from the normal. In the usual case, shoulder movements of small extent are utilized to control the locking and unlocking of the elbow, or the opening and closing of the grip. The actual energy for the arm movements themselves is provided by springs within the arm. After practice, the control movements become quite automatic. It is apparent, then, that the higher animals, particularly man, are a good deal more plastic than amphibians in the reorganization of CNS patterns. The limits must be established by further research.

THE CLASSIFICATION OF MOVEMENT

In the first part of this chapter, we were concerned largely with physiology. It is now necessary to turn our attention to the analysis of motion from a more psychological point of view. Here our problem is to develop ways of analyzing the movements of intact organisms which will permit us to classify like movements together and, thus, simplify the task of description.

There are very many ways in which movements can be classified. We may distinguish between arm and leg movements, for example, or between movements involving the fingers and those involving the shoulder. Motion study in industry uses a classification which divides any continuous act into "**therbligs,**" examples of such components are transport loaded, transport empty, positioning, grasp, and so on. These are not very useful to the scientist since they do not represent a consistent set of categories, even though they may provide a convenient shorthand for recording the movements made by workers. An even more serious objection is that therbligs are not really genuine components of movements — they do not provide a meaningful analysis.

Kinesiology, a field of investigation especially related to physical education, has attempted the analysis of movement. In general, the results are of interest from the point of view of joint rotations, particular muscles involved when a given joint is moved, and so on, but the analysis has limited psychological value. The organism probably has preferred ways of doing things which represent more natural movements than others, but these preferred ways are discovered best by the study of the organism as a whole.

As a matter of fact, a really good analysis does not exist. The classification to be presented in the next paragraphs is a very general one. but has the virtue of calling attention to some important aspects of movement which might otherwise be overlooked. It will be seen that movements may differ in terms of three sets of general criteria, and these are outlined below.

The Manner of Initiation. Movements differ in being initiated reflexly or voluntarily. For our purposes, a **voluntary movement** is one which is not directly related to a particular external stimulus and which ordinarily can be announced beforehand by the mover. These terms thus represent the extremes of what is really a continuous dimension. Some movements such as tendon jerks are at one extreme; going to the library or other planned acts are at the other. This dimension is not a trivial one and it can be demonstrated that reflexive and voluntary movements of the same muscles differ in some ways. For example, the reflex is faster than the voluntary response and is very much more difficult to inhibit. After considerable practice, voluntary movements may become so routinized as not to involve attention. In fact, it is commonplace knowledge that too careful attention to one's movements may upset them rather badly. Even a well-learned skill such as playing the piano from memory can disintegrate under such circumstances. There is no evidence, however, that such well-routinized acts involve different nervous structures from acts which require attention. What is implied is that once a well-integrated set of neural patterns is established interference from competing patterns leads to upset of the precise timing necessary for smooth action. Unfortunately, very little information is available concerning the neurology of serial action and so we can say very little indeed about the nature of the interference.

In the practical situation, some consideration must be given to the problem of making action as natural as possible. That is, it is desirable to find out what movements are toward the reflex end of our reflex-voluntary continuum. You can illustrate this point for yourself. Simply hold the two hands in front of you with the elbows close to the sides. Then try moving them from side to side as rapidly as you can in the same direction. Now move them in opposite directions. The latter will seem easier. Indeed, one of the rules of efficient work is that the hands should move in opposite directions simultaneously. The general problem of naturalness of movement patterns is one which is challenging for future research.

Muscular Balance during the Movement. It is convenient to classify movements into three categories depending on the balance of contraction between agonist and antagonist occurring during the activity. The first type we shall call **fixed.** Perhaps it will seem strange to classify motionlessness as a form of movement. The reference here, however, is not to mere relaxation, but to the maintenance of position through the fixing of a joint by active muscular contraction. A simple example is the holding of a stick which you are engaged in carving. Such movements will exhibit tremor and drift. In general, fixed movements are rather tiring, and holding an object is usually done better with a mechanical appliance. In industry, an attempt is ordinarily made to eliminate " fixed movements " where possible.

A second type of movement is called **controlled.** In a controlled movement, the agonist works against a load imposed by the antagonist. Most movements are of this type. One reaches for his pen, or a hammer, or places a tool in position for work. This type of movement can probably best be understood by contrasting it with the third, or **ballistic** type. In ballistic movement, little opposition is offered by the antagonist and the moved member swings almost as a pendulum. In normal walking, the leg describes a ballistic movement in its forward swing. In the purest case, a ballistic movement is initiated by a sudden contraction and the member continues under its own momentum. Needless to say, a pure ballistic movement is very rare, but movements partaking in part of ballistic characteristics will be found in skilled acts such as appear in sports or some manual skills. The proper technique of pounding nails approaches a ballistic movement. The common error made by the amateur is to attempt to " steer " the hammer, which simply results in stiffening the arm with resulting loss in both strength and accuracy. The emphasis upon " relaxation " and " follow-through " to be found in athletic coaching bears upon this point also. Here the idea is to keep those muscles relaxed which are not participating in the act, while the follow-through is evidence that the desired selective relaxation has actually taken place. No one who has ever seen a really good golfer hit a drive will ever believe that he is relaxed while he is doing it, and he is not. But only the proper muscles are contracted, and so he achieves free, ballistic-like movement.

It is obvious that a great many, if not the overwhelming majority of, industrial jobs have no place for such " free-wheeling " movements. Controlled movements appear in various forms, such as positioning, the setting of controls, filing, and many others. Also, one may distinguish between rectilinear and circular controlled movements, as might occur in cranking; and between single and repetitive movements. Sometimes these movements are subject to control by visual feedback, sometimes they are not. A typical example of control by visual feedback is what is called " tracking." In a military setting, this involves keeping a gun aimed at a moving target, and in the laboratory involves keeping a pointer centered on a wavy line which is carried past a viewing window on a moving tape. Such experimental conditions permit highly precise analyses of the time lag, rate of movement, and so on, of responses to various types of change in the line to be tracked. So far, however, these analyses have been rather restricted and lose significance in other than tracking situations.

The Manner of Termination. We shall consider three general ways in which movements may be terminated. First of all, and most frequently, a movement is terminated by the contraction of opposing muscle groups. One reaches for a pencil, lifts food to his mouth, or points at an interesting object. In all these examples the motion is stopped by muscular action. Such termination is also typical of the tracking movements which we discussed briefly under the previous heading. All of these examples are of controlled movements, but ballistic movements are sometimes terminated in the same way. An example would be properly struck octaves on a piano — the movement should be ballistic, but still terminate before the keys are crashed against the felt pads beneath them. Rapid repetitive movements such as tapping also can be of this nature. The forward swing of the leg in walking is a very familiar example of a ballistic movement terminated by muscular action.

A second way in which movements may be terminated is by the passive stretching of ligaments and muscles, and internal friction. A really thorough-going ballistic movement is of this nature. A good golf swing terminates very much in this way, as does a free swing with a baseball bat.

A third mode of termination is an external block. The movement stops because it comes up against something. A hammer blow is a very good example of a movement terminated by an external block. In such movements there may be considerable shock to the moving part and this can prove tiring unless somehow cushioned. A hammer handle, for example, should have some spring to take up the shock of the blow. Often the inept or inexperienced performer may — in hammering, boxing, and the like — " pull his punches," which simply means that he has terminated his blow in the wrong manner. Instead of letting the movement run its course and hit the external block with full force, he shies from the impact and permits opposing muscles to slow the movement before its termination.

Alternative Approaches to the Classification of Movement. The foregoing classification at least has the advantage of suggesting the wide range of problems to be encountered. There are other ways of approaching the problem of classification, however, and these give such dissimilar results that they cannot be ignored. At the present stage of research on human movement there is little if any translation possible from one set of categories to another. We shall consider, therefore, some of the alternative approaches because of their value in suggesting problems which might otherwise not be appreciated.

Single and Serial Action. It is often convenient to distinguish between acts which involve a single movement and those in which a succession of movements is involved. To take a very simple case, consider the difference between tapping a telegraph key once, as in a reaction time experiment, and tapping the same key repetitively. Sometimes a serial action involves more than the repetition of a single movement pattern, as, for example, when a pianist plays a rapid octave run or a complex pattern of notes. Sometimes, also, external control is partly responsible for the nature and timing of the serial acts, as when a pianist " sight reads " an unfamiliar composition. In this latter case, the conceptualization of how serial action comes about is not too difficult — we can apply our servomechanism analogy to it very nicely, since striking each note leads to looking at the next, and so on across the page. But accounting for a sequence of acts in which each act is the cue to the next is actually not as simple. Neurologically, we really know absolutely nothing about how a series of acts can come about — how, for example, one can recite the alphabet. This remains as a most intriguing problem for the neurophysiologist.

The Analysis of Individual Differences. Since the study of individual differences has been very fruitful in suggesting a set of dimensions for the description of mental abilities measured by paper-and-pencil tests, it is only natural to ask whether or not a similar approach might be useful in the field of motor ability. We can, for example, give a battery of motor tasks to a large group of subjects and analyze the results in order to find which abilities go with which others. The results of such studies have been somewhat astonishing. Movements involving the same musculature in very much the same way are not necessarily related. For example, speed of reaction as measured by lifting the hand from a telegraph key is not closely related to the speed with which one can tap with the same key. Also, there seems to be no general factor of motor ability which runs through all tasks. This conclusion is bolstered by the fact that it has been very difficult to find motor tests which will predict skill in either factory work or athletics. All in all, the results of this approach — the analysis of individual differences — have been far from clear. A few categories of motor ability suggested by one study are as follows:[12] (1) speed of single reaction; (2) finger, hand, and forearm speed in restricted oscillatory movement (e.g., tapping); (3) forearm and hand speed in oscillatory movements of moderate extent (e.g., two and three plate tapping tests); (4) steadiness or precision; and (5) skill in manipulating spatial relations (which is probably a perceptual category). A later study has suggested still another set of categories.[13] Unfortunately, none of these categories is unambiguous and more research will have to be done before complete confidence can be placed in them. At the present, they are suggestive of an approach, not a perfected set of results.

Applied Experimental Psychology. During and since World War II, research on the design of equipment from the point of view of the characteristics of the human operator who must use it forced a consideration of the psychological analysis of movement. The categories actually used could scarcely be claimed to represent an enduring and consistent set. Since considerable interest is being shown in this field of research at present, a sample set of categories, with illustrative tasks, is given below.[14]

1. Positioning. This refers to the task of placing some bodily member, usually the hand, at some given point in space. Positioning may be visually aided, or it may be "blind." Positioning may also be either free, as when one is asked to point straight up, or it may be restricted, as when one must move a lever or turn a knob to a given end-point. Much of the research on positioning has been aimed at the proper placement of the various controls in an airplane cockpit.

2. Repetitive Movements. Repetitive movements may be of several types, tapping and cranking motions being the most important. Research on repetitive movements has included studies of the proper operation of telegraph keys, and the cranking motions made in tracking a target by means of a hand wheel.

3. Load Bearing. Some work has been concerned with the efficient carrying of loads. For example, in carrying a load, should one balance it on a hip, place it on the head, or what? Actually, a yoke across the shoulders is least effortful.

4. Fixation. This refers to the holding of the body, or some part of the body, in a given position. Here we encounter problems of sway and tremor.

5. Direction of Movement. Some studies have concerned themselves with speed and accuracy as a function of the direction of arm movements in relation to the body. It is important in some industrial settings, for example, to decide at what angle the hands should move across a work surface for maximum speed and precision. Such problems arise frequently in assembly operations.

There are still other aspects of applied experimental psychology which cannot be fitted into the above categories. Some of these aspects have to do with highly specific problems. An example is **prosthesis** research, particularly as related to the design of artificial legs. The problem here is that of constructing an artificial leg that will simulate the movements of a real leg, for cosmetic reasons, if no other. Before the engineering problems can be tackled, it is necessary to analyze the movements of real legs. This has been done by means of photography and devices which register the varying pressures of the foot during the walking movement. From these studies it has been possible to outline the components of the walking movement and to design legs which perform quite well, not only for walking but for climbing up and down stairs and steps, and arising after falling.

MEASURING THE COST OF MOVEMENT

It is not fitting to conclude a discussion of movement without some attention to the measurement of its physiological cost. The most common way of obtaining this cost is by measuring oxygen consumption or carbon dioxide output. The apparatus may be made reasonably portable and actually carried on the subject's back during wheelbarrow work, and so on. A typical set of results is given in Table 18-1.

Table 18-1

The oxygen consumption in c.c. per horizontal kilogram meter for different methods of carrying loads of two different weights in different positions. Adapted from Bedale, E. M. Comparison of the energy expenditures of a woman carrying loads in eight different positions. *Indus., Fat., Res., Bd., Gt. Brit.*, 1924, No. 29B. Table XVII, p. 22.

Position	Weight (pounds)	
	20	50
On one hip	.121	.141
In back pack	.118	.137
On head	.109	.131
In tray on sling	.103	.131
In tray on hands	.100	.142
In bundles	.099	.122
On one shoulder	.090	.122
From yoke	.082	.097

Such an analysis can be useful in assaying the cost of physical work, but it must be remembered that other factors are important in the applied situation. For example, sitting still, though not very energy consuming, may be more fatiguing than some activity which requires more movement.

Another direct way of measuring the cost of movement is by measuring bodily heat output. Since the oxidative processes in the muscles are the source of body heat, any change in heat production represents a change in muscular activity. Unfortunately, the complication of the apparatus required for the measurement of heat output, plus the necessity of confining the subject in an enclosed chamber, has made this method impractical for most research. The lactic acid content of the blood is also an indicator of muscular activity, since lactic acid is a product of muscular activity. However, for significant amounts to accumulate in the blood would require such a state of fatigue as to make this index useless in the ordinary situation.

Suggested Readings

1. Ruch, T. C., " Motor Systems," S. S. Stevens Ed., *Handbook of Experimental Psychology*. New York: Wiley, 1951.
2. Fitts, P. M., *Engineering Psychology and Equipment Design*, Chapter 35 in *ibid.*
3. Sperry, R. W., *Mechanisms of Neutral Maturation*, Chapter 7 in *ibid.*
4. Chapanis, A., Garner, W. R., and Morgan, C. T., *Applied Experimental Psychology*. New York: Wiley, 1949 (Chapter 10, " How we make movements")

19 Cortical Dynamics and Integrated Behavior

In the preceding chapter we assigned to the cerebral cortex the major role in the integration and control of movement in the mammal. The same situation holds for all aspects of mammalian psychological functioning. As the result of encephalization, the cerebral cortex has become involved in everything that the organism does. Later, we shall consider more fully how this statement applies to the control of the metabolism, normal and pathological, of the organism. Here we shall be concerned with the cerebral cortex itself, with its interrelationships with other CNS structures, with its nutrition, and with its manner of physiological function.

FUNCTIONAL AREAS OF THE CEREBRAL CORTEX

There is no question whatever that different regions of the cerebral cortex differ in function. But they do not differ in the manner suggested by phrenologists, whose maps of the psychological functions of different brain areas can still be seen from time to time at county fairs or in city amusement zones. From Figure 19.1, which reproduces one of these charts, it will be seen that the various supposed psychological faculties — amativeness, combativeness, and so on — are assigned to specific cortical areas. Presumably, taming a combative person could be accomplished by cutting out the combative area. We now know that a more likely result would be visual disturbance. Reaction against such an approach must not, however, lead us to think of the cerebral cortex as one homogeneous mass of tissue, since it certainly is not.

Neocortex and Allocortex. The cerebral cortex may be divided into two major subdivisions: (1) the **neocortex**, or " new " cortex, and (2) the **allocortex**, or " other " cortex. The cellular structure of the allocortex is different from that of the neocortex and, in general, the allocortex developed earlier in the course of evolution. If one looks at Figure 19.2, which is a lateral view of the left cerebral cortex, he will see nothing but neocortex — the allocortex is visible in the intact brain only in small part on the ventral surface. Although there are several structures which make up the allocortex, knowledge of their names is not essential to the present discussion.

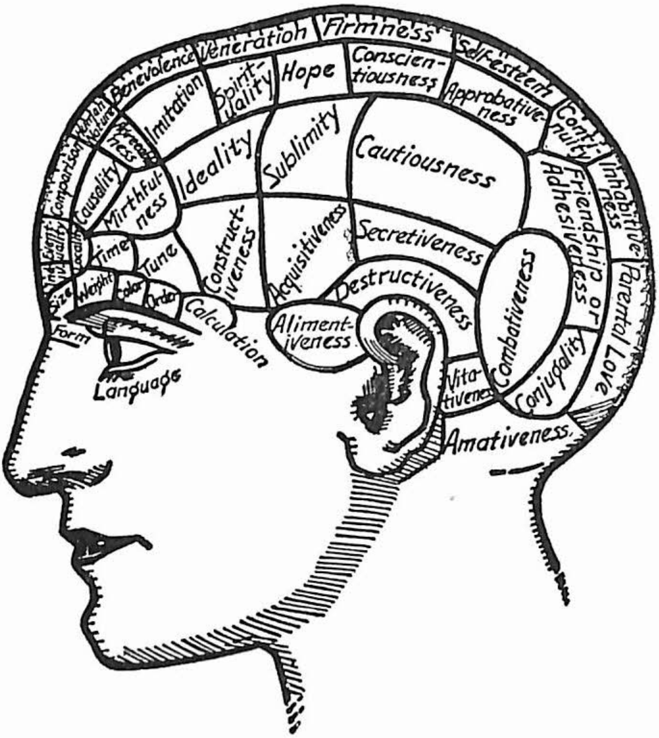


FIG. 19.1. Localization of psychological functions and capacities as alleged by phrenology. (From Hepner.[1])

Because the allocortex maintains an intimate relationship with the olfactory system, it has been thought of as the "smell brain." Actually, modern research has assigned it considerably greater importance in the over-all activity of the organism than this would imply. Stimulation of the allocortex is most likely to yield autonomic responses, or somatic responses of the organism, such as attacks in apparent rage which are, at least superficially, of an emotional nature. These responses are mediated through the connections which the allocortex maintains with the hypothalamus and other subcortical structures. Operations removing parts of the allocortex may greatly reduce the ferocity of some animals. The allocortex receives sensory impulses from the viscera. Therefore, it is to be thought of as being intimately concerned with the internal activity of the organism. It is not exclusively so concerned, however, since rage or other emotional patterns evoked by stimulation of the allocortex involve somatic activity (such as biting, running, and vocalizing). Also, autonomic activity can be evoked by stimulation of the neocortex. But, in general, the identification of the allocortex with internal activity is useful.

Subdivisions of the Neocortex. The neocortex may be divided grossly into lobes, or more finely into areas identified by their cellular structure. Figure 19.2 shows both varieties of division. The frontal, parietal, occipital, and temporal lobes are convenient terms for gross localization, but it must not be thought that each represents a homogeneous functional area.

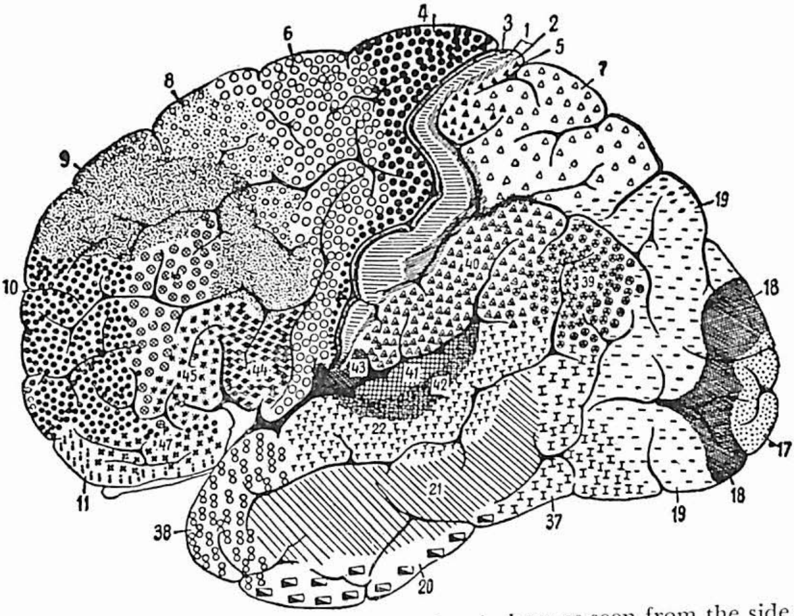


FIG. 19.2. Cerebral cortex of the left hemisphere as seen from the side. The lobes are comprised of the following areas: 1, frontal lobe, area 4 and the areas anterior to it; 2, parietal lobe, area 3 and the areas posteriorly through area 39 and ventrally through area 40; 3, occipital lobe, areas 17, 18, and 19; and 4, temporal lobe, the remaining areas. (From Herrick.[2])

The subdivision of the neocortex on the basis of cellular pattern is called **cytoarchitectonics.** We shall return to the cellular structure of the cortex in a later section. The numbered areas in Figure 19.2 represent distinguishable cytoarchitectonic regions and these have considerably more importance in identifying functional areas than does the division into lobes. We shall use the numbers for several of the more important areas in subsequent paragraphs.

Thalamic Connections. All regions of the neocortex receive connections from the thalamus and in all instances send fibers back to the thalamus. The precise functional significance of the two-way pattern of connections is still in doubt. Some of the projections from thalamus to cortex subserve sensory functions, as has been pointed out in the chapters on the senses. Perhaps some of the downward connections, from cortex to thalamus, underlie the phenomena of attention by permitting cortical activity to determine to some degree what thalamic activity shall be permitted to influence the cortex.

In the sensory systems, the connections between thalamus and cortex are fairly precise. In other regions of the cortex, especially those which we shall discuss under the general heading of association areas, the connections may be rather diffuse. Probably they serve a general function of arousal or alertness.

Sensory and Perceptual Functions. The primitive origins of the cortex in lower animals were related to sensory systems. Sensory and perceptual functions are still extremely important cortical functions, despite the fact that the portions of the cortex not directly related to sensation have shown the greatest enlargement in recent evolution.

The senses of vision, audition, and somesthesis have well-known neocortical areas. Vision has its primary cortical representation in the occipital lobes, specifically in area 17 as shown in Figure 19.2. Most of the visual area is actually hidden from view, since it lies on the medial surfaces of the hemispheres. Areas 18 and 19 also are concerned primarily with vision, but do not receive fibers directly from the visual system. Stimulation of these areas (17, 18, and 19) produces, in the waking human patient, visual sensations such as flashes of light, colored disks, and dancing lights.[1] Removal of area 17 produces blindness in which the person has, at most, only vague visual awareness.

Audition has its primary area in the temporal lobes (Chap. 11). Stimulation of areas 41 and 42 produces ringing and buzzing in one or both ears, or may change the quality and intensity of perceived sounds in peculiar ways.[1] Sometimes the patient reports dizziness as though vestibular function were also represented here, but removal of these temporal lobe areas does not result in impaired balance. Bilateral removal of the auditory areas results in deafness.

Somesthesis is represented in areas 3, 1, and 2 of the parietal lobes. Stimulation of this primary projection area gives rise to sensations of tingling, pressure, or kinesthesis, and occasionally of cold and warmth. The regions of the body to which the sensations resulting from direct cortical stimulation are referred are represented in

Figure 19.3. The topography corresponds to that of the motor cortex. Damage to the somesthetic areas results in defects of somesthesis. If the loss is so severe that the patient cannot distinguish objects by touch or manipulation, he is said to suffer from **agnosia.** Sometimes there is an upset in the patient's awareness of himself, even to the extent that he denies ownership of an arm or a leg. This upset in the **bodily image** is of importance because perception of oneself underlies the development of the concept of the self, or **ego.**

Other primary sensory areas are less well-known. Taste is probably localized in the somesthetic region for the face.[2] Olfaction appears to be at least largely an allocortical sense. There is some possibility that pain has cortical representation in the frontal lobes, probably in the region of area 11.[3] Perhaps visceral sensation also has representation there.

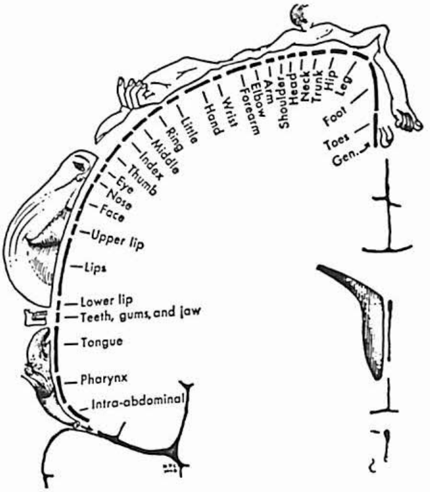


FIG. 19.3. Sensory homunculus. The right side of the figurine is laid upon a cross section of the hemisphere, drawn somewhat in proportion to the extent of sensory cortex devoted to it. The lengths of the underlying black lines indicate more accurately the comparative extents of the representation in the cortex. (From Penfield and Rasmussen.[5])

Motor Functions. An entire chapter (18) has been devoted to the control of movement and the topic will not be elaborated here. However, it is important to note that in man the dependence of motor function upon the cerebral cortex is greater than in other animals. Stimulation of the motor cortex of a lower mammal may result in very little movement, which is confined to the head and upper extremities. But in man, movement may be localized and precise upon stimulation of area 4. It is to be re-emphasized, moreover, that motor fibers arise from many areas of the cortex outside the so-called motor areas, so that changing patterns of cortical function in many areas influence the activities of the organism.

Association Functions. There are two large areas of the human cortex which are not directly concerned with sensory or motor functions. These are the frontal association areas, sometimes called the prefrontal area and the parietal-occipital-temporal (POT) association area. These areas have sometimes been called "silent areas" because damage to them does not result in specific loss of sensation or motor function.

The frontal association area has been shown to be concerned, in monkeys, with the ability to delay response and to combine elements of a situation which are not simultaneously present. Partly on the basis of these results and partly on the basis of clinical study of persons having tumors of the frontal lobes, it has been suggested that the frontal association areas are concerned with the higher intellectual functions. The frontal association area has received considerable recent attention because of the operations of **prefrontal lobotomy**, or separation of the prefrontal areas from the thalamus, and **prefrontal topectomy**, in which small bits of prefrontal cortex are removed. A surprising result of these operations is the reduction in the importance or insistence of pain. Intellectually there is probably some reduction in planning ability, and a reduction of anxiety and muscular tension occurs in some cases, but carefully controlled studies of topectomies have revealed little or no objectively measurable impairment as the result of the operations.[4] Whether there is a genuine "frontal lobe syndrome" in man is still in doubt.[5]

The POT association area is concerned with visual, auditory, and somesthetic perception and, apparently, is involved in what may be called "perceptual memory." Stimulation of portions of this region may induce images of familiar objects or scenes.[5] Visual recognition seems to be affected by damage to this association area. In addition, peculiar affective changes have been ascribed to damage to the temporal region, and in human patients irritation to the temporal lobes may give rise to what is called "temporal lobe epilepsy," in which affective components are combined with momentary blurring or loss of consciousness. The precise mechanism is obscure. Severe interference with the use of language, called **aphasia**, also may occur following damage to the POT association area. The particular type of difficulty, whether of auditory or visual word recognition, for example, will depend upon the locus of the causative damage.

Our fund of knowledge about the function of the association areas is still rather slight. No doubt one reason for this is that the association areas, being the last cortical structures to develop, may subserve psychological functions of such complexity and delicacy that our tests are not sufficiently subtle to reveal defects in them.

Integration among the Lobes. The facts of perception show very clearly that many senses must cooperate to produce awareness of an object or event. The discussion on page 132, in which flavor was found to be dependent upon the simultaneous action of many senses, will serve as an illustration. And one not only comes to blend simultaneous sensory material, he also comes to expect certain patterns. A really sour chocolate drop, a hot cake of ice, a racing auto which passes with no sound whatever — any one of these would startle because past experience has led us to expect certain associations of sensory material.

How do these blends come about? How does the behavior of the human organism somehow reflect the integration in the CNS of diverse information, both past and present? At one time it was thought that the long connecting tracts within the cerebrum might be responsible. But there is no evidence that any lobe to lobe tracts have any important function. Even putting numerous vertical cuts in the cortex itself has no demonstrable effect. In brief, we do not know how over-all integration comes about, or where it comes about.

MICROSCOPIC STRUCTURE OF THE CORTEX

An understanding of the different approaches to the question of how the cerebral cortex functions depends upon a minimum knowledge of the fine structure of the cortex. Eventually, of course, explanations of function must be compatible with the facts of microscopic anatomy, although, as has already been seen in regard to the long " association " tracts, function cannot always be deduced from anatomy alone. Nevertheless, anatomy does set the limits to our flights of theoretical fancy.

Supporting Cells. The neurons of the cortex are virtually imbedded in a mass of supporting cells, called, collectively, **glial cells** or **neuroglia.** These are of several types, which are not of concern to us here. The interrelationships between glial cells and the neurons are not clearly known. Perhaps the relationship is partly a nutritive one. One set of observations indicates that the glial cells actually contract individually at regular (5 minute) intervals, giving the associated neurons a massage in the process.[6] This could, of course, be quite important to the normal metabolism of the neurons. In general, however, one becomes aware of the neuroglia only when it runs astray, producing tumors of the CNS.

Cytoarchitectonics. The term " cytoarchitectonics " refers to the cellular structure of an organ, in this case the cerebral cortex. Needless to say, the fine structure of the cortex is extremely complicated, particularly in man where the fundamental mammalian structure has had added to it very large numbers of neurons with very short axons. Figure 19.4 shows a section of human cortex from the motor region, with only the cell bodies of neurons stained. The exact appearance depends upon the region of the cortex from which the section is taken and the numbered regions of the cortex that we have been using in this chapter are based on the study under the microscope of such small differences.

Of particular interest is the close packing of neurons and glial cells. When we consider that those spaces which do exist among the cells are filled with fluid, and that the cortical tissue is extremely

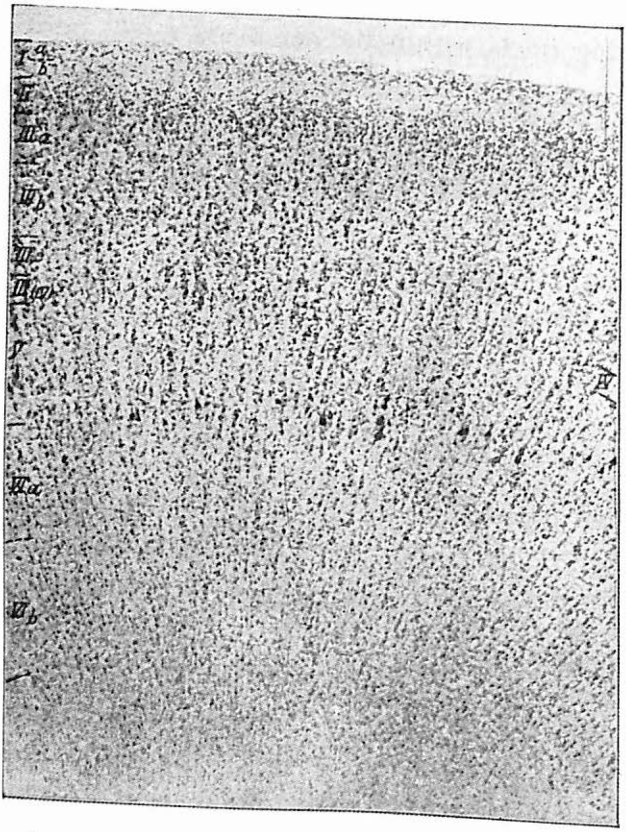


FIG. 19.4. Section of the human motor cortex (area 4) showing the neuron cell bodies. (From Economo.[11])

soft, we can understand why some theorists look upon the functioning of the cortex as involving mass electrical effects, rather than conduction through isolated neurons only.

Central Synapses. In addition to the usual type of synapse that has been discussed before, what is called **neuropil** is found in the CNS. Neuropil refers to a mass of intertwined axons and dendrites which serves to permit diffuse interrelationships among neurons. Figure 19.5 illustrates a mass of neuropil. It is difficult to imagine that connection among neurons involved in neuropil is precise. We shall return to this problem later in this chapter. As far as the transmission of impulses is concerned, there is every likelihood that central synapses behave much as do other synapses.

METABOLISM OF THE CEREBRAL CORTEX

As in the case with any living tissue, the CNS, including the cerebral cortex, must carry on the fundamental processes of metabolism. However, there are various particular ways in which a given tissue can metabolize and there are specific things which can affect the metabolism of a given tissue. In the case of the brain, we shall be interested in its utilization of oxygen, its source of food, and the way in which circulation affects its activities.

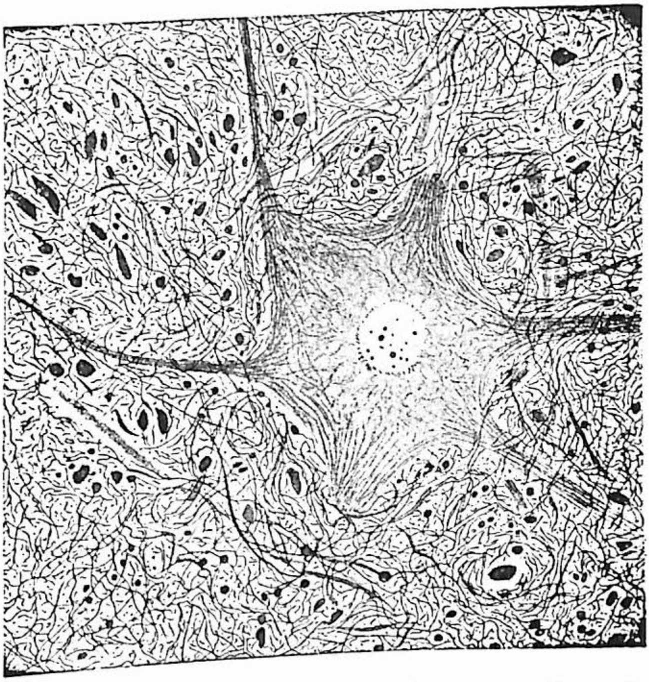


FIG. 19.5. Neuropil around a spinal neuron. (From Ranson and Clark.[12])

Cerebral Oxidation. In common with the other tissues of the body, the energy for cerebral activity is basically derived from the oxidation of foodstuffs. Complete oxygen deprivation results in very rapid breakdown of CNS activity, permanent damage resulting in a matter of a few minutes in the adult. The newborn can tolerate oxygen deprivation without harm for a longer time, which must be the result of a different pattern of CNS metabolism. In general, the different divisions of the adult CNS show metabolic rates directly related to their recency in evolutionary development. The cerebral cortex thus has the highest metabolic rate, that is, it uses oxygen most rapidly, while the spinal cord has the lowest rate. As might be inferred, the structures requiring the most oxygen are the most easily affected by oxygen deprivation or by drugs which interfere with oxidation.

If interference with oxidation lowers CNS activity, it may be asked whether the reverse is also true. Are alterations in activity reflected in changed CNS oxidation? The answer is, in general, yes. Cerebral oxidation is always depressed if the organism is in a comatose condition, no matter what the initiating cause. On the other hand, increases above the awake, resting level are not so dramatic. It is just barely possible to demonstrate an increase in oxygen consumption by the entire body when resting is compared with doing mental arithmetic and much of even this small increase must be due to slight increases in muscular tension. The waking brain is spontaneously active and mental arithmetic no doubt represents more a change in pattern of activity than an increased level of activity.

Too high an oxygen pressure is also a disadvantage. The respiration of the central neurons is interfered with, resulting in an upset in function. Sometimes hyperventilation (overbreathing) is used to test the stability of the CNS, since it will lead to abnormal EEG's, but this effect is more likely due to the reduction in carbon dioxide supply, which in turn changes cerebral circulation. It is well to avoid thinking that all changes in cerebral activity can be explained by changes in brain oxidation. For example, patients with so profound a psychosis as **schizophrenia** show normal cerebral metabolic rates.

Cerebral Foodstuffs. In discussing cerebral foodstuffs, we shall confine ourselves to those foods which are uniquely important for the brain. The basic supplies of proteins, carbohydrates, fats, minerals, vitamins, and so on, must be available to the brain cells just as they must be to other cells of the body. However, cerebral metabolism has points of difference which need consideration.

In the preceding section we have discussed the oxygen requirements of the CNS. The question now arises as to what is oxidized. There is no doubt that the overwhelmingly important source of energy is **glucose.** Glucose is a simple sugar derived largely from the carbohydrates and partly from the fats and proteins in the diet. Glucose is made continuously available in the normal organism, since it is stored in the form of glycogen or " animal starch " primarily in the liver and striated muscles. A serious fall in blood sugar level, called **hypoglycemia,** can have considerable consequences for the CNS. In general, however, blood-sugar level is maintained by the action of the sympathetico-adrenal system. In addition, somatic utilization of glucose is inhibited by ACTH and corticosterone, so that under extreme conditions what glucose there is, is available to the CNS. Insulin lowers blood glucose (by increasing its utilization by somatic tissues) and, as a result, large doses of insulin can result in reduction in brain activity, coma, and death.

The action of hormones in influencing cerebral metabolism is probably indirect, through their control of blood-sugar level and oxidation of glucose elsewhere in the body. There is evidence that neither insulin nor corticosterone will pass the so-called " **blood-brain barrier** " and, thus, cannot act directly on the neurons.[1]

Vitamin B_1 has been shown to be important to the complete oxidation of glucose, hence to normal nervous activity. Severe restriction of intake of this vitamin will give rise to upsets in metabolism in various parts of the CNS, as well as in peripheral neurons, and mental symptoms may result. For example, it has been reported that the production of severe Vitamin B_1 deficiency results in irritability, depression, inability to tolerate pain, and anxiety. It is interesting in this regard to note that many of the symptoms of acute alcoholism are ascribable to Vitamin B_1 deficiency.

Cerebral Circulation. We shall consider the cerebral circulation as consisting of two divisions: (1) the blood vessels supplying the CNS and (2), the cerebrospinal fluid. Since the cerebrospinal fluid is derived from the circulatory system in the brain, we shall consider this system first.

As is the case for other bodily tissues, the CNS is well supplied with arteries and veins, the details of which are of no importance to us here. Also, in keeping with the usual pattern of circulatory supply, the CNS contains capillaries connecting the arteries and veins, through the walls of which the metabolic supplies for the brain and spinal cord must pass in order to reach the tissue fluids and eventually the cells. If, therefore, a substance will not pass through the capillary walls, or is blocked at any other point before actual action on a cell, it will not affect cerebral metabolism directly. Taken collectively, these possible points of blockage are called the " blood-brain barrier." Examples of substances which will not pass this barrier are glutamic acid and insulin.

Since the vascular circulation brings to the brain all its metabolites, including food and oxygen, changes in circulation may be expected to have demonstrable effects on cerebral activity. Complete blockage of circulation to a given brain area results in prompt cessation of activity and very rapid irreversible breakdown. As might be expected from our previous statement concerning the progressive effects of oxygen lack, the newer brain structures can tolerate the cessation of circulation and, hence, oxygen and food supply, much less well than can the older. The cerebral cortex deprived of circulation will cease activity in about 10 seconds and will show irreversible changes in a very few minutes, whereas the corresponding times for the medulla are about 2 minutes and 7 or 8 minutes. It is easy to see, therefore, why a person revived after a fairly prolonged period of circulatory failure may show normal vital reflexes, but exhibit dementia and personality changes. Cases where a patient has been " brought back from death " by heart massage, for example, are fairly frequently reported.

Whether more subtle changes in circulation are of importance in influencing cerebral activity is not completely clear. If a general lowering of blood pressure occurs, as the result, let us say, of shock, fainting is likely to occur. There is evidence in man, moreover, for autonomic control of the blood vessels of the brain itself.

Other events also can influence the cerebral circulation. For example, the cerebral blood vessels will dilate under the influence of carbon dioxide, thus increasing cerebral circulation locally in regions of activity. As a specific instance, stimulation of the eye with light produces definite increases in circulation in the lateral geniculate bodies and occipital lobes, because their increased activity produces CO_2.

The cerebrospinal fluid is evolved almost entirely from masses of capillaries lying within certain ventricles of the brain, called **choroid plexes.** This fluid is similar to the serum of the blood (that is, blood without the blood cells), but is not identical to it. An important function of this fluid is the protection of the CNS, which is afforded partly by the damping it provides to shock and jiggling, and perhaps more importantly by the fact that the effective weight of the brain is very much reduced since it is floating in the fluid. Except for pathological conditions, as when the fluid is overplentiful, little is known about the subtle influence of the composition of the cerebral spinal fluid on brain functioning.

PROBLEMS OF CEREBRAL DYNAMICS

So far in this chapter we have examined the general organization of the cerebral cortex and have considered its sources of food and energy. It is now necessary to turn to topics which are at once more specific — they involve a consideration of how the neurons themselves are interrelated, and more general — they involve speculation about the kinds of scientific model which will best describe how the cortex works. In general, we shall see that speculation has taken two basic directions. In the first of these, cortical activity is to be understood in terms of the interrelationships among individual neurons, operating through specific synapses, with integration being derived from the timing of activity. In the second direction, cortical activity is to be understood, at least partly, in terms of field effects — the individual neurons are less important than the patterns of activity themselves, and mass effects of electrical and chemical fields are basically important.

Nerve Impulse Theories. The approach to the understanding of cortical action which we shall term collectively " nerve impulse theories " assumes that the activity of central neurons is the same as that of peripheral neurons. In other words, a neuron in the CNS, including the cerebral cortex, has one basic function, the conduction of nerve impulses. It may be said, therefore, that a given neuron has but two modes of existence. It is either conducting a nerve impulse or it is not. Whether it is or is not, at a given moment, depends upon the activity of the neurons which discharge at its incoming synapses. In turn, its activity helps determine the activity of neurons further along. When we consider the billions of neurons that are present in the CNS, it is apparent that the combined action of these neurons can occur in an almost infinite variety of patterns, both in space and in time. The patterns of the moment determine the perceptions, thoughts, and activities of the organism.

It is well to remember that to say that the action of the cerebral cortex is to be understood in terms of specific neuronal activity does not mean that each neuron has a special, individual function. There is not, for example, one neuron for each memory, or even one chain of neurons for each association. Evidence from brain injury in man and from experimental ablation in animals is all against such a view. What the theorist has in mind is that the *patterns* of neuron activity are important, but that these patterns are patterns of nerve impulses, not of electrical or chemical fields. Sometimes the analogy of the digital computer is used. Such a device consists of a very large number of vacuum tubes, connected in a complex circuit. Each tube has two possibilities at a given moment — it can be conducting a current or it can be silent. Thus, the analogy between a neuron and a vacuum tube is an easy one to make. Digital computers are the most complex devices we have at the present and this complexity is derived from a relatively simple principle — a tube which has only two possible ways of behaving. Perhaps this explains the popularity of this analogy among the more imaginative neurophysiologists and psychologists. Unfortunately, no one really knows whether the computer model is a good one; and discussing the model is no substitute for research on the brain itself.

Field Theories. The field theories suggest that the neurons of the cerebral cortex can influence each other in ways other than at synapses. If a given neuron is active, for example, it may change the excitability of neurons near it. In this way, we may imagine, a group of active neurons would create a strong field of influence, partly because of the electrical phenomena which are part of their nerve impulses, and perhaps partly because of the chemical changes which are also part of the nerve impulses. In one form of this type of theory, the fields of activity underlying perceptual phenomena have a physical shape very much like the perception. This conception is obviously much better suited to visual patterns than to, say, taste blends.

There is good evidence that neurons can influence each other outside of synapses. Activity in a given neuron will alter the threshold of a neuron near and parallel to it. Effects of activity extending over a millimeter or two have been observed in insect ganglia. Added to this functional evidence, field theorists can point to the anatomical fact that there are a multitude of tightly packed small neurons in the cortex and to the existence in the CNS of areas of the apparently diffuse intertwining neuron processes we have called neuropil. Such regions do not seem to provide the precise synaptic relationships implied by strict nerve impulse theories.

Nerve Impulse and Field Theories Contrasted. At the risk of serious oversimplification, let us consider how nerve impulse and field theories would account for the discrimination of a triangle from a circle. Perhaps this choice of example is unduly favorable to field theory. Discriminating sweet from sour would be much harder to explain in field theory terms. For the simple field theory, this discrimination is very easy. The triangular stimulus throws a triangular image on the retina and this shape is retained in the neural activity right up to the striate cortex. That is, the pattern of cortical excitation is roughly triangular in shape. Similarly, the circle leads to a circular pattern of activity in the cortex. How and where these two patterns are *compared* is somehow seldom discussed. Given the patterns, discrimination and appropriate behavior are considered to follow.

Now, before leaving the field theory part of our example, it is well to point out that this extreme form of **isomorphism** (triangle leads to a triangular pattern of activity) is not indispensable for field theory. The pattern in the cortex could be anything at all, provided each figure gave rise to a different pattern with some constant way of predicting point by point the pattern from the stimulus. However, most field discussions have implied the form of isomorphism which we have used in our example.

Nerve impulse theory cannot find so apparently simple an explanation as can field theory. Two more complicated suggestions can be considered here. First of all, it is possible to imagine that each organism must build up during the early years of its life specific aggregates of neurons for the different shapes. If a triangle and a circle can be discriminated, it is because triangles and circles have, through learning, come to arouse specific aggregates of neurons in the visual association areas. Such a conception of how the cortex works is based largely on studies of animals reared in darkness and of people operated on for congenital cataracts later in life. Although the evidence is somewhat ambiguous at present, that of a necessary learning period predominates.

Another nerve impulse approach states that each perception in our example gives rise to a pattern of impulses involving the whole visual cortex, much as a pebble cast into a pond sets up a pattern of wavelets covering the entire surface of the water. These patterns may be considered to be partly spatial, partly temporal, but certainly not in any direct way copying the physical outline of the stimulus. They are patterns of nerve impulses, depending upon the arrangement of synapses and the timing of impulses in the cortex, not patterns existing as electrical or chemical fields in which the individual neurons are not basically important.

Which general approach will hold up best in the light of future research we cannot be sure. Our discussion has left out the refinements of each. If it were necessary to hazard a guess at this point, the best would be that both the field and nerve impulse theories have elements of correctness, and the cerebral cortex is sufficiently complex to allow several modes of integration to proceed at once.

The Source of the Electroencephalogram. The electroencephalogram has been referred to briefly in Chapter 4. It is possible to record changes in electrical potentials of the brain by using electrodes attached to the scalp. The EEG ordinarily represents fluctuations in potential above and below an unknown baseline, which sometimes are referred to as " brain waves."

When one analyzes a series of EEG records, he finds that the greatest amount of the energy changes are of rather low frequency — 12 cycles per second and below. In most relaxed subjects who are awake but not mentally occupied, the predominent rhythm is about 8 to 12 per second, depending on the individual. These are called **alpha rhythms,** and are illustrated, along with some other rhythms, in Figure 19.6. Alpha rhythms are most strongly recorded over the occipital regions of the head. **Beta rhythms** are faster, about 25 or more per second, and are more characteristic of the motor areas. When the subject slips off into sleep or unconsciousness, the rhythms slow down, first to the three per second **delta rhythms,** and finally to very slow random waves. All these are shown in Figure 19.7. The **kappa rhythms,** an additional variety which have about the same frequency as the alphas, have been reported from electrodes over the tips of the temporal lobes, but their full significance is not known. The EEG represents an informative, though gross, indicator of cerebral activity. At the moment we shall be concerned primarily with the alpha rhythm. This is characteristic of the waking but relaxed subject. When the subject is stimulated with light, a loud sound, or other stimulus, or when he thinks about something, his alpha rhythm will be " blocked." Figure 19.7 illustrates a typical record of **alpha blocking.**

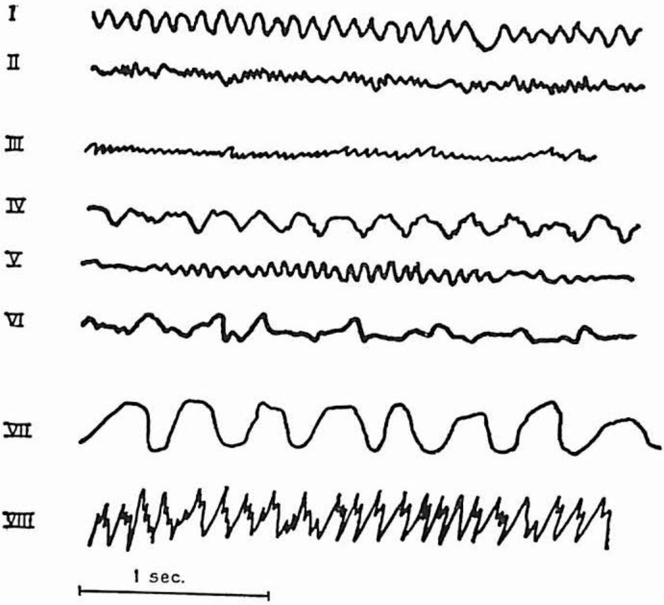


FIG. 19.6. Some varieties of wave found in the human electroencephalogram. I, alpha; II, beta; III, gamma; IV, saw-tooth; V, spindles; VI, random; VII, delta; VIII, spikes. (From Crafts, et al.[15])

The possible sources of the alpha rhythm are of at least two general kinds. These kinds are of the same sort as the division between nerve impulse and field theory approaches discussed in the paragraphs above. One suggestion is that they arise from the summated nerve impulses of synchronously firing cerebral neurons. According to this view, blocking represents the breaking up of the synchrony by the establishment of patterns of excitation in the cortex which cannot be detected in the EEG. It has further been suggested that the maintenance of this rhythmical, synchronous discharge depends upon the interconnections between cortex and thalamus, so that a positive feedback relationship is established. Thus, volleys of impulses make the circuit from thalamus to cortex and back again in continuous fashion so long as outside interference does not occur. Probably this view is incorrect and the rhythms are not dependent upon these interconnections, since isolated cortex can show spontaneous rhythmical activity.

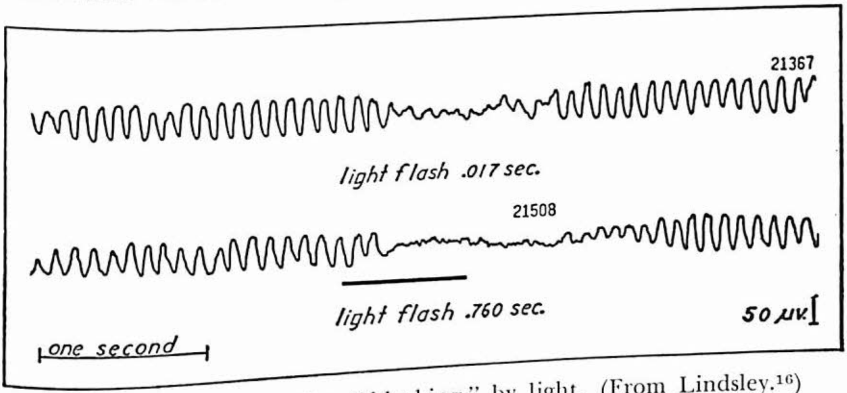


FIG. 19.7. Alpha rhythm "blocking" by light. (From Lindsley.[16])

Another approach considers that alpha rhythms do not represent impulses at all, but rather that they arise from the rising and falling of potential waves either in the cortex as a whole or in the membrane potentials of the cortical neurons. Recent work indicates that this view is probably correct. The alpha rhythm, and probably other cortical recordable rhythms, is not a summation of synchronous nerve impulses but represents over-all fluctuation in potential. Precisely how this affects the activity of the cortex is not certain, but the suggestion that it represents a waxing and waning of cortical excitability has considerable plausibility. If this is true, the cortex, at least in the visual region, is maximally excitable at about 1/10-second intervals and there is some evidence that this is indeed true.[9] Whether or not this suggestion will withstand the scrutiny of future research cannot at the moment be decided.

LEARNING

The nature of the physiological changes underlying learning is a fascinating problem which has been the subject of considerable speculation, but virtually no empirical progress. There are really two general problems. First, what structures are necessary for learning? In order to sharpen the problem, we shall assume that the CNS is the locus of learning and our discussion will be confined to it. Second, what changes occur in the necessary structures when learning takes place? This question we shall consider after our discussion of the first.

CNS Structures Necessary for Learning. The consideration of the structures necessary for learning is complicated by the number of different types of learning situations which can be studied. Of these, the classical conditioned response is the simplest, instrumental conditioning the next least complicated. About their neural loci we can state a few general conclusions. There is no doubt that conditioning can take place in the decorticate animal. Thus, the cerebral cortex is not essential for simple conditioning, although it does participate ordinarily when it is present. Whether or not spinal conditioning can occur is a matter of dispute, with the evidence probably indicating that conditioned responses mediated by the spinal cord are either very rare or nonexistent.

Discrimination habits, such as choosing a light versus a dark alley, represent considerably more complication. In man, the cortical sensory areas seem to be essential even for the simpler types of discrimination, such as light versus dark. In the rat, however, such is not the case, for a light-dark discrimination can be learned in normal time after removal of the visual cortex. Animals having color vision, the primates particularly, need the cortical visual areas for color discrimination. The cerebral cortex is also essential for pattern vision. Analogous results have been obtained for the other senses. The cerebral cortex is essential for subtle discriminations, the subcortical centers being sufficient for grosser discriminations such as intensity — in animals other than man, at least.

Maze learning has been extensively studied in the rat and a considerable variety of ablations performed. Since it has been found that loss of maze learning ability is more nearly related to how much cortex is removed than to what parts of the cortex is removed, we may conclude that maze learning is so varied a task that the rat uses whatever cues are available, thus involving a great many cortical areas. Removing various senses by ablations of the receptors also interferes with maze learning and we may tentatively conclude that the removal of the cortical areas acts largely by reducing the variety of available cues.

When we discussed the functions of the frontal lobes we had occasion to mention the effects of lesions in this region on problem-solving behavior. Where the elements of a situation must be integrated, it would appear that the frontal areas are of importance. In the rat, manipulation of complex problem-box material is affected by brain lesions much as is maze learning.

It should be pointed out that studies of the effect of brain lesions on performance in learning situations must take account of at least four factors. First, sheer modifiability of function is a property of a large part, if not all, of the CNS. It is not likely that the effect of brain lesions on maze learning, for example, is to be considered as loss in sheer modifiability, as witnessed by the normal learning time for simple discriminations. Second, brain lesions may have a serious effect upon the motivational status of the organism, either reducing its energy or upsetting other of its internal conditions. Third, removal of portions of the cerebral cortex reduces the resources of the animal for integration — for putting together the cues from several senses and utilizing them to reach successful solutions to problems. This would seem to be an important aspect of the results obtained in the study of maze learning in the rat. Finally, and this is of considerable importance for problem-solving behavior, removal of parts of the cortex seems to reduce the range of performance of an animal. Its repertoire of responses is reduced and it becomes less variable. Thus, the probability of discovery of a correct solution is decreased.

Neural Changes in Learning. It has been thought for some time that the most likely locus, in the microscopic sense of the word, for the neural changes involved in learning is the synapse. As learning progresses, new neural patterns must be established. This implies that new relationships must be established among the activities of the neurons. The most obvious point of change is at the synaptic junctions among neurons.

If we assume for the sake of discussion the accuracy of the hypothesis that the synaptic relationships change during learning, the problem of accounting for such changes remains. This problem is really double. First, how do the synapses change so that a neuron now no longer fires one that it fired before, or now can excite one which it could not excite before? Secondly, and no less difficult, how can the outcome of an activity work to influence synaptic change? Why is the response which reaches the goal learned, the blind alley entry eliminated?

Actually, there is no accepted theory of how synaptic resistence is altered in learning. No doubt the changes are biochemical — which says nothing whatever except that they are in the organism. Perhaps when a neuron excites another, their membranes, which make contact at the synapse between them, grow more alike chemically and make the synapse more intimate. But this is sheer speculation since no one has ever demonstrated such changes. Besides, this answer begs our second question since, if mere simultaneous action underlies learning, the effects of the response cannot be a direct factor in learning but must be accounted for by some other principle.

Our second question has no answer. Perhaps, indeed, it is not a legitimate question (a point which would take us far into learning theory if pursued) and the effects of a bit of behavior have no direct action on learning. But both common sense and experiment incline one to think that they do. At the moment, no suggested explanation is sufficiently plausible to warrant inclusion here.

Suggested Readings

1. Jeffress, L. A. (Ed.). *Cerebral Mechanisms in Behavior: The Hixon Symposium.* New York: Wiley, 1951.
2. Morgan, C. T., and Stellar, Eliot. *Physiological Psychology.* New York: McGraw-Hill, 1950. Chapter XXVI.

20 | *Motivation*

The Definition of Motivation. A fundamental assumption in science is that every action has a cause. Thus, in the biological sciences, for each act of an organism an antecedent cause is assumed. Sometimes the cause of response, in man for example, is essentially external and the cause and effect relationship can be verified by many witnesses. Sometimes the cause is internal and known to the responder only; yet his explanation is accepted as credible by others because they have had similar experiences. Sometimes, however, the cause of a response is internal but unknown, both to the responder and to others.

When a given organismic act follows a given change in the external environment, it usually is said that stimulation has occurred. When a given act follows a change known to have occurred within the organism, one sometimes speaks of internal stimulation, especially if it is known that recognized interoceptor cells have been activated or if the behavior is brief, as in a reflex. In the absence of specific information about internal stimulation, to account for other than reflexive behavior, psychologists usually employ another term — **motivation.** Thus, in a constant cage environment, if one animal drinks and another eats — or in the library, if one student studies and another day-drams — we usually say that the differing behavior is due to different motivation. Motivation, then refers to conditions or states within an organism that cause persisting behavior; and in the above examples we might speak of the **motive** to drink, the motive to eat, the motive to study, or the motive to day-dream. It will be evident that such usage is usually based on inference and that it follows from the assumption that behavior is caused. The study of motivation is an investigation of the nature of the internal and variable determinants of persisting behavior and of the mechanisms by which they exert their effects.

Some Comments on Terminology. Man has always been interested in causes. In his attempts to describe why he and his fellow creatures do what they do he has come to use many words in addition to stimulation and motivation. "**Drives**" and "**needs**" are perhaps the most common; but others frequently met are "urges," "tensions," " need-states," " need-presses," " **releasers,**" " **sensitizers,**" " energizers," " sets," " instincts," " dispositions " and " habits." For man, in particular, there is still another group: " desires," " wishes," " wants," " plans," " purposes," " interests," " likes," and " dislikes." We cannot discuss the usage of all these terms in one chapter. All that we can hope to do is present those that, in our opinion, are most useful in describing behavior from a physiological point of view and to qualify or restrict some of the others.

As a term in psychology, motivation is often confused with five others: energy, habit, emotion, instinct, and need. In the usage we employ, a motive may serve to release energy in effector cells but it itself contributes little energy. A motive may serve to induce the behavior that becomes established as habitual (i.e., a habit) and it may arise in new form from the habit (as in drug addiction, for example); but habit is behavior and any motivation resulting from such behavior should be considered separately. This is also so with emotion. In the next chapter we shall define emotion as behavior. In common with any organismic act, it sets up a new condition within the organism which may serve to induce subsequent behavior. The other two terms cited in this paragraph — instinct and need — receive more extended comment in the following paragraphs.

Instinctive Behavior. " Instinct " as a noun and as an explanation of behavior has practically disappeared from modern psychology. Many writers refer, however, to **instinctive behavior.** By it they usually mean a pattern of activity that is common to a given species and that occurs without opportunity for learning. They may differ about how stereotyped or how complex the activity must be if it is to be called instinctive; but most will agree that it involves more than single reflexive acts, that the sequence of acts may be somewhat variable, and that it depends primarily upon inherited neurophysiological mechanisms. Most will agree, also, that in species which show marked learning ability, instinctive behavior patterns either disappear or are masked by learned behavior. Thus, few writers today emphasize instinctive patterns in man, or even in the anthropoid apes. For this reason, we shall devote little space to the subject, although it is important in any study of comparative psychology. It involves such varied reaction patterns as those of flight or attack, food taking and hoarding, hibernation and migration, mating and nest building, and the complex sequence of patterns having to do with care of the young. In so far as these activities are common among higher organisms we shall consider them in subsequent paragraphs — not in terms of patterns for given species, but in terms of their motivation.

The Concept of Need. Although some psychologists use " need " and " motive " as synonyms, you shall see that we make a distinction between them. Since they are related, however, and since we all use " need " so frequently in our thinking and conversation, a consideration of biological needs forms one appropriate approach to the subject-matter of this chapter.

First, we want to make it clear that need has no physical existence; it is entirely a conceptual matter. We speak, for example, of a house needing paint or needing a new roof. In this case, "need" refers to a desire or purpose to protect the house, or to protect the furnishings of the house, from the weather. If one were an intended suicide, his first need would be for a means of death. If he were to choose a tall building and jump from it, he would not need wings, or a net, or a parachute. If his fall were accidental, however, obviously one of these things would be quite necessary to him. The need relates to the goal. His goal of death is one thing; his goal of continuing life is another.

Need has no meaning except in terms of a stated or implied goal. But it would seem that it has two distinct uses: (1) we speak of organisms needing food if they are to continue to live, or (2) we speak of organism *x*, which has not eaten for a week, as needing food. With engines we speak (1) of automobiles needing gasoline if they are to operate, or (2) of automobile *x*, which has exhausted its supply, as needing gasoline. The first statements here are generalizations; they are statements of fact but they denote necessary conditions for some end. The second statements are specific. They apply to a given engine or organism and, more particularly, to a specific condition or state of that engine or organism. Condition or state of need, then, is different from need when used alone; so we distinguish this meaning by the hyphenated term, **need-state.**

If need-states are capable of inducing behavior they are motives. But it is important to realize that they may exist and still not induce adjustive behavior. For example, we spoke in Chapter 15 of the cretinous child who was deficient in thyroxin. Such a deficiency does not elicit behavior. Instead it serves to reduce it; and a need-state for thyroxin cannot be regarded as a motive. Even more illustrative is the difference between our need for oxygen (O_2) and our need to eliminate carbon dioxide (CO_2). Both are necessary for the integrity of the organism but, as we shall see later, the addition of O_2 in animals occurs largely as a result of accumulated CO_2. In other words, a need-state for O_2 has little motivational significance; but excessive CO_2 in the blood stream is an important motive. It leads us to breathe and thereby to both eliminate CO_2 and acquire O_2.

Releasers and Sensitizers. Two other terms previously cited deserve particular attention. They are used most frequently, perhaps, in discussions of instinctive behavior; but they can be of more general value. One, **sensitizer,** usually refers to some internal organismic condition which serves to lower the response threshold for a given behavior pattern or to induce it. The other, **releaser,** usually refers to rather specific external stimulation which, in the presence of a sensitizer, will " release " a rather specific behavior pattern.

Mating in subprimate female mammals furnishes a good illustration of the manner in which releasers and sensitizers together determine behavior. The advances of males typically are spurned except for a brief period associated with ovulation. Then the visual and other stimuli afforded by a male, or even another female, will release the mating responses characteristic of the species. The sensitizer (or motive) here has been shown to be hormonal in character. When hormonal levels are appropriate, the female becomes " receptive " to the male and is said to be " in heat," or in **estrus.**

Much of man's behavior, also, is dually determined by sensitizers and releasers. Consider your own reactions to a drinking fountain: (1) often you pass it without drinking; (2) sometimes it automatically releases drinking responses; (3) and sometimes when you are strongly sensitized you seek it even from some distance, but you do not go through the movements of drinking until you reach the fountain. It should be evident that releasers are of little significance as behavior determinants in the absence of motivation, but also that the character of motivated behavior is determined, in part, by external stimuli which may serve to release either unlearned or learned behavior.

Some Models of Motivation. Let us turn now to some models that should serve to emphasize the points we have made. A common model of behavior in psychological textbooks is shown in Figure 20.1. This merely says that ◯, the organism, is influenced by an external

$$S1 \longrightarrow \bigcirc \longrightarrow R1$$

FIG. 20.1

stimulus, $S1$, and makes a response, $R1$. If behavior were that simple, we should have no need for the concept of motivation.

Other models, however, illustrate its importance. Figure 20.2 involves the same external stimulus as in Figure 20.1 but the response

$$S1 \longrightarrow \textcircled{x} \longrightarrow Rx$$

FIG. 20.2

is now different. We may say that state x within the organism has served to modify the response, or, preferably, that state x has, in the presence of S1, served to elicit a new response. Moreover, in Figure 20.3, state x alone induces the new response, when S1 either remains constant, is absent, or the organism is blocked from receiving it.

$$S1 \diagup \textcircled{x} \longrightarrow Rx$$

FIG. 20.3

In Figure 20.2, state x is the sensitizer or motive, and $S1$ is the releaser. In other words, S1 releases Rx if ○ is primed or sensitized by state x. But in any study of motivation the essence lies in Fig. 20.3. Here, x within the organism alone elicits Rx. When we come to understand all the x's within the organism, we shall understand motivation and we shall better understand behavior.

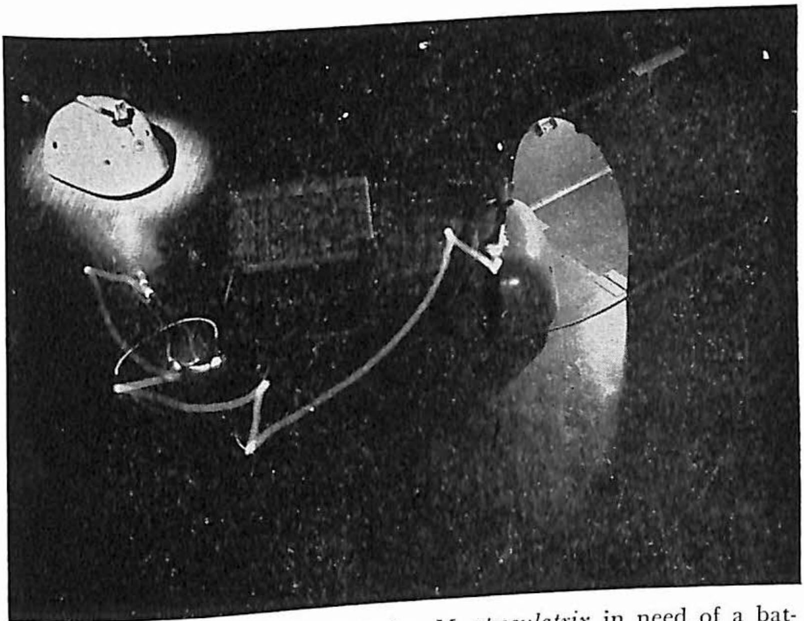


FIG. 20.4. The mechanical tortoise, *M. speculatrix* in need of a battery recharge. The time exposure shows the path taken as it avoids an obstacle while approaching the bright light of its hut. (Photograph by Desmond Tripp, Ltd. and by courtesy of Dr. Grey Walter.[1])

Several investigators have attempted to further this understanding by constructing machines which behave like human beings or at least like infrahuman organisms. For example, one British scientist has designed *Machina speculatrix* — a turtle-shaped apparatus which we use for our last model [1] (see Fig. 20.4).

This machine has two amplifiers, two relays, two electric motors, two batteries, and two " receptors " — a photoelectric cell which is sensitive to light and an electrical touch-plate which is responsive to physical contact. The circuit is so arranged that " exploratory " movements are typical in darkness and moderate lights are " attractive," but bright lights and physical objects are " repulsive." Thus, the machine demonstrates both approach and avoidance behavior to external " stimuli." When its batteries need recharging, however, the bright light in its garage is no longer avoided but is approached. As it enters the garage, its " feeding " electrodes engage with the battery-charging circuit and a relay turns off the power for its motors. The machine then remains motionless until its batteries are charged — that is, until its " need-state " has disappeared. Then, it is again repelled by the bright light and physical contact of its garage and departs to continue in the activities of which it is capable.

We see, then, that it is possible to put specific adjustive acts into a machine if we provide suitable irritable mechanisms and suitable control circuits connected with locomotion. If we really want to make a machine self-adjustive, we should have to put into it either a large number of specific acts and need-state irritabilities or provide, in some manner, the capacity for learning.

Most submammalian species continue to live and continue to reproduce themselves because their inherited structures permit adjustive responses to need-states. In mammals, and particularly in the higher primates, the greater capacity for learning minimizes the importance of inherited adjustive response patterns. But the external or internal determinants of behavior in a machine, in amoeba, or in man have much in common. They are represented in simple fashion in *M. speculatrix*. But even amoeba is more complex. As pointed out in Chapter 2, it and all organisms react to chemical, thermal, mechanical, and electrical stimulation; and higher animals react to these and others — both external and internal. We already have considered the major external determinants of behavior (Chaps. 6–14) . Now we consider some of the internal ones in man and other animals.

The Concept of Drive. As previously stated, the terms " need " and " drive " are frequently employed in psychological discussions of motivation. We have discussed the concept of need and introduced the term " need-state." We now define **drive** as any unlearned motive. Since many of the motives common to a given species are associated with general biological needs (i.e., necessities for individual integrity and species continuation, regardless of species) , drives often are need-states. But need-states are not always drives. Table 20-1 serves to reemphasize the difference and to present some of the more general biological needs in comparison with their associated drives.

It should be pointed out that, at present, there is little consistency among psychologists in the manner of designating different drives. It is common, for example, for an author to speak of the hunger drive, the drive for water, the sex drive, and the drive to eliminate. But the first refers to a sensation; the second refers to a **goal** or **incentive;** the last to a form of behavior; and "sex," used alone, is ambiguous to say the least. In part, this lack of consistency is due to inadequate information about the nature of drives; in part it is due to concentration of attention on the motivated behavior itself or on its apparent objective — that which when achieved results in termination of the behavior. When we know more about drives, it will be easier to direct our attention to them and to classify and describe them systematically. In the meantime, we can only make a step in that direction. The right hand column of Table 20-1 and the discussion that follows represents such an attempt. We begin by discussing in some detail the drive of water deficit and the behavior it elicits.

Table 20-1. Physiological Needs and Drives of Organisms

Needs	Associated Drives
I. *For Functional Integrity*	
A. *Agents or conditions to be added*	
Water	Water Deficit
Food	Food Deficit
Vitamins and Minerals	Some Specific Deficits
Oxygen	0
Hormones	0
Adequate Temperature	(See Below)
B. *Agents or conditions to be eliminated*	
Extreme Temperatures	Thermal Deficit Thermal Excess
Waste Products of Metabolism and Digestion	CO_2 Excess Urine Excess Feces Excess "Fatigue"
Other Sources of Tissue Damage	Excess Foreign Matter Pain
II. *For Species Continuation*	
Reproductive Functions	Sexual Excitement
Care of Offspring	Prolactin

The Drive of Water Deficit. Water is the major component of living tissues. In man it constitutes about three-fourths of the body weight. As living cells lose water their functions are impaired and in organisms with a circulatory system the blood volume decreases and leads ultimately to circulatory failure and to death.

For man on the desert, for example, it is estimated that a water loss equal to 20 percent of his body weight is sufficient to produce death. But long before this amount of water deficit has occurred, his functioning has become impaired. He experiences difficulty in muscular movements including breathing; he loses his desire for food and becomes nauseated; he becomes tired to the point of exhaustion; and his initial sensations of thirst become overlaid with thoughts and even illusions and hallucinations about water.

One of the first signs of such a state of progressive dehydration would be the sensation we call thirst. For many years it was thought that the dryness of the mucous membranes at the rear of the throat could be regarded as the source of the drive for water — as the motive for drinking. Several lines of evidence indicate, however, that we must look elsewhere for at least a portion of the drive: (1) denervation of the throat does not alter the total amount of water drunk by experimental animals over given periods of time; (2) drugs which enhance salivary secretion, and thus keep the throat moist, do not alter the quantity of water drunk; (3) with a depletion of sodium chloride (common salt) from the body, as by excessive sweating, the urge to drink is decreased, although water deficit has increased and the mouth becomes excessively dry; and finally (4) the taking of food, even moist food which does not dry the throat, creates an additional urge to drink.[2]

Such evidence does not mean, of course, that sensations from the throat are unimportant in determining drinking behavior. In fact, one experiment illustrates rather conclusively that some mechanism connected with the act of drinking (perhaps in the throat) can, in effect, measure the amount of water needed to restore physiological equilibrium. After a dog had been deprived of water for some time, and a known water deficit produced (as measured in terms of weight loss), the water was replaced directly into the stomach through a tube. If allowed to drink immediately after this artificial replacement, the dog drank enough to equal its original deficit, but if the opportunity to drink was deferred for 20 minutes drinking did not occur, and if deferred 10 minutes some drinking occurred but not enough to make up the original deficit. We may conclude that both drinking and amount of water taken are influenced by some mechanism associated with drinking and that this motive may be removed either by water which passes through the throat only or by water placed directly into the stomach; but in the latter case time must be allowed for the water to be assimilated. That this assimilation is necessary was demonstrated by another test in which a surgical opening (a fistula) was created in a dog's stomach so that the water he drank could be removed immediately after drinking. Under such conditions drinking occurred again within a few minutes.

Other experiments have demonstrated that the tissue state that initiates drinking and determines the amount drunk is probably one of cellular dehydration. For example, the injection of strong salt solutions or other chemicals that cause loss of water from body cells results almost immediately in thirst and drinking, while conditions that produce cellular hydration inhibit drinking.

But even though cellular dehydration be the basic physiological condition, much in drinking behavior remains unexplained. One investigation suggests that the answer is to be found in a portion of the anterior hypothalamus where the physical changes in a group of nerve cells, when in a dehydrated state, initiate the nerve impulses which induce water-searching and drinking behavior. If these interpretations are substantiated by further research, the drive of water deficit will be describable as a specific form of stimulation in a specific area of the hypothalamus.

We should not overlook, however, that sensations from the throat serve in man, and probably in many other animals, to supplement the underlying drive. Since both would normally occur prior to drinking behavior, as learned behavior patterns develop, either alone would serve to elicit that behavior. Moreover, in so far as states of increased muscular tension had regularly accompanied such learning, such states also could be expected to elicit learned drinking behavior. Thus, our occasional drinking behavior when not " thirsty " and when certainly not in a state of water deficit can be explained. We shall have more to say later about tension as a motive. For the present it is sufficient to point out that motives that induce drinking behavior are not confined to pure water deficit.

The General Drive of Food Deficit. It is well known that man and other animals react selectively to certain foods but that at times they will eat almost anything. Selective eating will be discussed in the next section. Here we consider the general state of food deficit which occurs when an organism has not eaten for some time or has eaten in amounts inadequate for its sustenance. We are apt to say that it is " hungry " and attribute concurrent behavior to hunger sensations, but such a statement is incomplete. Although the sensations which we call hunger or hunger pangs, and which arise in part from extreme contractions of the muscles of an empty stomach, undoubtedly do aid in inducing eating behavior, they are no more necessary for it than is the sensation of thirst for drinking behavior. For example, the entire stomach can be removed in man and replaced by the upper intestine but he will continue to eat normal amounts and feel the urge to eat. Similar results occur after **vagotomy,** an operation sometimes performed on patients who have severe ulcers of the stomach. Although such an operation reduces stomach activity and eliminates sensation from the stomach, the patients eat normal amounts of food. Similar findings have been reported for experimental animals. We are forced to conclude that the drive of food deficit is more than hunger sensation from the stomach.

Other experiments suggest that the drive is some factor in the blood stream. If the blood of an animal who has been starved is injected into the circulatory system of one which is well fed, stomach contractions are intensified. If the reverse experiment is performed, stomach contractions are inhibited.

The search for this factor in the blood, which apparently determines both stomach motility and eating behavior, has not yet proved fruitful. A logical guess was blood-sugar level but research here has been indecisive. Recent research suggests that it may be low blood levels of certain amino acids, alone or in combination with low blood sugar.[3] Other experiments suggest that, whatever the blood factor may be, like the water deficit drive, it acts via the hypothalamus. For example, lesions in the lower and central regions of the hypothalamus will lead to voracious eating. Animals so prepared will eat most of the time and soon double or triple their body size. On the other hand, stimulation of the posterior hypothalamus decreases stomach contractions and eating behavior; and certain anterior hypothalamic lesions completely eliminate both. It seems clear, then, that some factor in the blood acts on anterior hypothalamic nuclei to produce both stomach contractions and eating behavior.

Before terminating this section on general food deficit, one unique experiment should be described. It involved conscientious objectors during World War II who submitted to a deficient diet for 24 weeks. It is significant in that it demonstrated under controlled conditions the wide-spread effects of food deficit. The subjects became fatigued, had little energy for any sports or work, and were easily irritated. Gradually they lost interest in practically everything but food. They dreamed night and day of the next meal or of past feasts. Conversation lagged but, if it occurred, it usually dealt with foods or things associated with food. A favorite pastime was looking at pictures of foods in magazines and reading cooking recipes. Some subjects even collected such material in scrap-books. Finally, as starvation progressed, they became more apathetic and morose. If they showed enthusiasm about anything it was about food.

While some of the change in behavior shown by these men can be attributed to their debilitated physical condition (the average weight loss was 30 pounds) this cannot explain their obsession with food. Apparently biological drives can determine much of the behavior of man if they persist unreduced, but we have few well-controlled experiments like this one to demonstrate the extent to which this is possible with a single need-state.

Some Specific Food-Deficit Drives. Although the drive discussed in the preceeding section probably is physiologically specific, the materials that will reduce or remove it are relatively nonspecific. Man can live for long periods on proteins, or carbohydrates, or fats; but in so doing he will develop cravings for other specific foods he has known and there is some evidence that his sense of taste will change. The same holds true if he is deprived of certain mineral salts, such as sodium chloride or calcium. After lack of either, or after injury to the adrenal cortices or the parathyroid glands whose hormones control the metabolism of sodium chloride and calcium respectively, he will consume large quantities of these salts and they taste " good " to him.

Most of what we know about such specific drives has come from experiments on food or mineral selection in rats. Some of the major findings are summarized: (1) some animals, but not all, will select an adequate diet over a period of time if given an opportunity to do so; (2) if caloric needs are increased or decreased by lowering or raising the environmental temperature, they will consume more or less carbohydrates; (3) during pregnancy and lactation, when certain mineral salts and certain foods are needed more than at other times, female rats usually will select additional amounts of the needed foods; (4) after removal of the pancreas, the adrenal glands, or the parathyroids, rats will alter their diets and choose foods or minerals that can be best assimilated or those of which the operation has deprived them; (5) habits of food selection in given environments will interfere with adequate food selection in those environments.

Such conclusions are interesting. They tell us that specific drives exist, but they tell us little about the nature of those drives. The psychologist who is responsible for much of this work believes that such specific need-states serve to lower the taste threshold for the needed substance. And he has shown that if the afferent nerves which mediate taste sensations are severed, rats no longer select needed salts. Another experiment has shown, however, that there is no apparent difference in sensory threshold to salt in normal and adrenalectomized rats. Perhaps it is just a matter of salt tasting "good" to a rat that needs it. We shall discuss this possible "sensory pleasure" in a later section.

Some Specific Needs without Drives. We already have indicated that not all need-states have associated drives and have mentioned in this connection both oxygen deficiency and hypothyroidism. Now we discuss them a bit more fully and add some others. Oxygen, of course, constitutes one of the major biological needs. We can live without it for only short periods of time and a deprivation of only 8 minutes will permanently damage nerve cells of the CNS.

The living organisms that we know, however, probably always have lived in an environment replete with oxygen. Hence, any which have been sensitive to oxygen lack have not been favored in evolutionary history. Today, few organisms react markedly to O_2 deficiency. Man possesses some tissues that are irritable to lowered O_2 in the blood stream (cf. Chap. 16) but they have little effect on his behavior beyond slightly increasing the rate of his breathing. He may be placed in artificial environments which are deficient in O_2, such as high altitudes or similar conditions in a low-pressure chamber, and, in the absence of instruction, he will not recognize his need-state. Instead, ironically, he will tend to see the world and himself through "rose-colored glasses." Even though his motor skills deteriorate and his writing becomes illegible, he will think that he is performing superbly. His judgment becomes impaired and he is unaware of it. In short, there is little oxygen deficiency drive.

In Chapter 15 we discussed the major hormones of vertebrate organisms and indicated their necessity for functional integrity. Deficiencies of any of them will drastically alter behavior and may even lead to death, but we have no evidence that drives are associated with any hormonal deficiencies as such. Behavior will be modified, as is the case with O_2 deficiency. Some deficiencies will produce specific drives of other sorts as we have seen in the case of deficient cortical steroids (salt craving), or parathormone lack (calcium craving), and some will serve to decrease sexual behavior as we shall see later; but no hormone lack directly serves as a drive because no such deficiency selectively activates the CNS.

Vitamin deficiencies are similar. Although many vitamins are known to be necessary for normal organismic function, none is known to provide selective drives. There is some evidence that deficiencies in some of the vitamins do produce preferences for those foods or liquids that normally contain them. For example, rats can learn to select foods or liquids that contain some vitamins of which they have been deprived and man develops cravings for fruits and vegetables even though his diet is adequate except for vitamins. The source of such specific urges probably depends on learning but, like others we have discussed, may depend on taste quality also. Much research will have to be completed before we can say more.

Drives Associated with Environmental Temperatures. The need to maintain an adequate body temperature, and its related drives, may be described in many ways. We might have spoken of a " drive for adequate temperature," but the concept would have been difficult to defend. Instead, in using the terms " thermal deficit " and " thermal excess " we more nearly approach the drive conditions and, moreover, provide a transition from deficit need-states to excess need-states.

All organisms (and machines, also) need appropriate temperatures for optimal performance. If temperatures become extreme, machines break down and, without temperature-sensitive circuits, do nothing about it. Organisms do something. They react to temperatures which are inappropriate for their optimal function. Thus, they react to both low and high temperatures by specific reflexes and by avertive movements. Some, in particular birds and mammals, have developed sensitive heat-regulatory mechanisms which permit a considerable range of adjustment without retreat to another environment. Although such reflexive mechanisms vary in different species they have three common factors: (1) control of heat loss at the body surface via circulatory and respiratory changes; (2) control of body surface temperature by evaporation of water, usually sweat; (3) internal heat production by alterations in muscular activity.

Heat loss and surface temperatures, of course, are closely related, but are essentially different in animals with ample sweat glands. For them sweating from most areas of the body is decreased in cool and increased in warm external temperatures, and the temperature of the skin and surface blood is influenced by the amount of sweat evaporation. Thus, when external humidity is low and sweat evaporation is rapid, greater surface cooling occurs and greater external temperatures can be tolerated. Panting serves a similar function, particularly in animals with few sweat glands; and the elephant, who apparently neither sweats nor pants, makes good use of his trunk as a unique sprinkler system.

The amount of heat loss at the body surface, however, is controlled largely by the amount of blood close to the surface, a function of the relative vasoconstriction or vasodilation in the small blood vessels and the rate of blood flow. The third mechanism, heat production, also is reflexive in its initial stages and involves increases or decreases in tension in striate muscles which produce heat as they contract. As external temperatures rise, relaxation occurs and overt activity decreases; as they fall, tension increases, the shivering reflex may occur, and overt activity usually increases.

The optimal environmental temperature for man with some clothing is approximately 72° Fahrenheit. Then his skin temperatures remain at stable levels and his internal (oral) temperature is about 98.6° F. With minor alterations in internal temperature alone, or in the environmental temperature with subsequent internal changes, his reflex mechanisms respond appropriately to maintain his optimal internal temperature. With more extreme alterations he engages in behavior which will continue, as long as he is physically able, until his optimal temperature has been achieved and he "feels comfortable." He moves to the shade or into the sun, he puts on or removes clothing, he turns on the furnace or the fan. But such motivated behavior is initiated by the same basic drives that activate his reflexive heat-regulatory mechanisms.

We know a bit more about these thermal drives than we do about most physiological drives, but the information still is far from complete. The drive we have called "thermal excess" seems to be a blood temperature higher than that which is optimal for a given organism, which activates temperature-sensitive nuclei in the anterior hypothalamus and results in the pattern of peripheral vasodilation, sweating or panting, and muscular relaxation. The pattern can be produced experimentally in an anesthetized animal (without the muscular changes, of course) by even slightly heating the hypothalamus; and destruction of the anterior hypothalamic nuclei destroys the pattern. It is unknown whether or not the temperature receptors of the skin contribute significantly to the response.

The thermoreceptors of the skin that respond to cold, however, probably are of major importance in eliciting heat retention and production. Not only are there many more of them than those that respond to warmth, (cf. Chap. 9), but the hypothalamus may be cooled without producing heat-retention responses. Since such responses are lost after destruction of posterior hypothalamic nuclei, it is assumed that the thermoregulatory center for heat retention and production is located there and that it is activated directly or indirectly by the discharge of afferent fibers associated with "cold" receptors in the skin. In some species, at least, it is probable that a portion of the activation is attributable to downward discharge to the hypothalamus from the somesthetic projection area of the cortex, because lesions there will interfere with heat retention.

We see, then, that we cannot speak of a single primary "drive for adequate temperature" but instead must speak of at least two drives, and possibly more. Until their precise nature is understood, we can think of (1) a drive of thermal excess, as being essentially an increased blood temperature that activates the anterior hypothalamus and effects heat loss, and (2) a drive of thermal deficit, as being essentially a central afferent discharge from cutaneous receptors that activates the posterior hypothalamus and effects heat retention and production. In man at least, however, we can speak of a learned motive to provide an adequate temperature. He prepares himself in many ways to tolerate extreme temperatures. Although thermal drives have seldom been used in learning experiments, it seems probable that many infrahuman animals also could become motivated, through learning, to engage in behavior that would serve to maintain an adequate temperature for them at a future time. Whether they normally do so is debatable.

Drives Associated with Waste Products of Metabolism and Digestion. A number of drives arise from the accumulation of waste products in the body. Often the elimination of excessive accumulation is largely reflexive in character but, if elimination is even temporarily prevented for any reason, it becomes an important determinant of behavior. Prominent among such drives are excess CO_2, urine, fecal matter, and "fatigue." The last is least well understood and probably most significant in determining our daily behavior, so we discuss it first.

Fatigue. **Fatigue (psychological),** as we shall employ the term at the moment, is a feeling or perception that usually increases with work-output and with time elapsed after rest or sleep. The underlying factor or factors that give rise to feelings of fatigue would be the drive or drives, but we do not know their nature. Some research suggests that a major factor in fatigue is the accumulation of lactic acid in muscle tissue and in the blood as a result of muscular contraction. Even if this proves true, we do not know the mechanism by which it induces rest (muscular relaxation) or sleep, and we do not even know that the drives and central mechanisms are identical for rest and for sleep. What is known about the central control of sleep is discussed elsewhere in this text (cf. Chap. 23) and will not be repeated here. We can say little more except to hazard the guess that the drive of "fatigue" will prove to be one or more factors circulating in the blood stream and will be found to determine the unconditioned responses of both rest and sleep.

Excess carbon dioxide. Carbon dioxide excess has already been mentioned in connection with respiration and the need for oxygen. In organisms with lungs, the major loss of CO_2 occurs there; and reflexive breathing is largely controlled by nuclei in the medulla oblongata of the brain stem which are irritable to even slight increases in CO_2 in the blood stream.

As metabolism continues in body cells, the oxidation of food is accompanied by the liberation of heat, CO_2, and water, H_2O, and other waste products. The latter will be discussed in the next paragraph, and heat loss already has been considered. Normally, CO_2 loss presents no problem and reflexive breathing both supplies us with oxygen and eliminates excessive CO_2. However, if this elimination is interfered with (as by holding the breath or by breathing air with greater than normal amounts of CO_2), rapid and deep respiration soon occurs with feelings of suffocation. If the condition is not self-imposed or cannot be terminated, greatly agitated behavior will appear, followed by unconsciousness, and finally death. In our normal environment, the drive of CO_2 excess is of little psychological significance. In an environment of greater CO_2 concentration, it would be an important determiner of behavior.

Water and other waste products. Water and other waste products of metabolism are largely filtered from the blood by the kidneys and excreted as urine. The drive of urine excess is a pressure phenomenon in the bladder and possibly other portions of the genito-urinary system. In lower animals, it operates reflexively at spinal levels; in higher animals, such as the dog, it may involve brain stem centers and induce searching behavior and particular postures which differ for males and females; and in man, in some cultures at least, rather complex behavior patterns may be elicited which depend in part on learned motives. The significance of the basic drive itself as a determinant of behavior is easily demonstrated when urination is prevented or voluntarily inhibited for some period of time. Under such conditions, as one unique experiment has shown, there is loss of ability to concentrate on other matters and a marked increase in muscular tension and restlessness.

Other waste products of metabolism and undigested materials accumulate in the lower intestines and stimulate pressure receptors in them that induce defecation. Here also the behavior is largely reflexive and is mediated primarily at spinal levels. But again, if prompt defecation is prevented or voluntarily inhibited, other centers become involved. Muscular tension and restlessness increase and widespread behavior changes occur. Thus, the drive of feces excess is of little psychological significance unless its prompt reduction does not occur.

Drives Associated with Other Sources of Tissue Damage.* All of the drives we have discussed can, under certain circumstances, produce tissue damage. We should give some attention, however, to other sources of tissue damage that may serve as drives. In general, they are of two sorts: (1) excessive foreign matter in the body, and (2) agents that cause pain.

Excessive foreign matter. As a drive this has received scant attention from psychologists, yet it may be as significant in determining behavior as some of the more commonly discussed drives. Witness the actions of a man who has eaten "something that doesn't agree with him," or who has gotten "something in his windpipe." The first may lie down, may search for an "anti-acid," may seek a place to regurgitate, or may even use an emetic. The second will engage in a fit of coughing and show other signs of agitation.

Matters that are foreign to the body may enter it at any of its orifices or by penetrating the skin. Pain fibers frequently are stimulated, but often the situation may be regarded merely as uncomfortable or distressing. The latter seem reducible to two factors — particular chemical irritants and excessive pressures. For example, gas in the stomach, or food between the teeth, primarily stimulate internal pressure receptors and elicit behavior even though no pain is involved. While over-distension of the stomach may be painful and may induce nausea and vomiting, the latter response patterns more frequently are produced by particular chemical irritants which typically are regarded as unpleasant but not painful. So with foreign matter in the lungs, such as the congestive accumulation with a chest cold. But in the trachea ("windpipe") where there are more pain receptors, or in the eye where there are even more, pain is more frequently a part of the reaction.

We shall not attempt to catalogue the various responses that are elicited by excessive foreign matter in various parts of the body. When they occur, they often dominate other behavior but they occur infrequently. One, the pattern of nausea, seems to be of some significance, psychologically. As was pointed out in Chapter 9, it also may be elicited by motion, via the vestibular apparatus. We refer to it again in the next chapter.

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* Excessive production of hormones could be considered here, or as a separate category; but, although excessive hormone levels alter behavior and may induce additional behavior and although they often cause tissue damage, they represent atypical states that fall outside the purview of the present text. The drive properties of such states as hyperthyroidism or hypergonadism should not be overlooked, however, and in a later section we shall mention the effects of normal variations in female gonadal hormones.

Agents that cause pain. Pain-producing agents are important determiners of behavior. They commonly are regarded as sources of tissue damage, but there are many exceptions — in particular, mild electric shocks that are painful yet do not harm the body. Moreover, if pain fibers are nonfunctional, extensive tissue damage may occur without inducing behavior. We are forced to conclude that it is the central volley from pain fibers which must be regarded as the drive to much avertive behavior — not the " need to prevent tissue damage." Since the subject is of particular importance in physiological psychology we shall consider it in some detail.

Pain as a Drive. In Chapter 8 we discussed pain as sensation. In the next chapter we shall regard pain as a primary emotional response. Here we discuss it as an important drive in human and animal behavior and emphasize the fact that the effect of stimulation is not necessarily unitary. It might be better if we had entirely different terms to designate the different aspects of the effect, but since we do not we shall have to be content with " the sensation of pain," the response of pain," and the " drive of pain."

Pain-producing agents may induce reflexive action that eliminates or averts them at once. As such they are of little more importance than the agents that induce a single cough or a single eye-blink. If they persist, however, the behavior persists and we speak of motivation. We might attempt to list all the agents that produce persisting pain and refer to them as the " drives " in avertive behavior, a procedure we have followed in this chapter when we have talked about specific deficits or excesses. Here, however, the important factor seems to be continued activation of pain fibers. So, as was the case for heat-retention behavior, we regard the central discharge of a group of afferent neurones as the drive.

As you know, pain fibers project to the diencephalon. Some reflexive avertive behavior patterns are mediated at spinal levels, but the variable and persisting organized behavior that continues as long as painful stimulation continues seems to be organized largely at the diencephalic level. The cortex may be unnecessary for its occurrence, but adds to its smoothness and direction. Further, there is evidence from prefrontal lobotomies in humans (cf. Chap. 8) that the frontal cortex contributes to reactivity to pain. After bilateral lobotomy the pain is still perceived but has lost some of its power as a drive. Thus, in man at least, the CNS mechanisms by means of which the drive of pain induces behavior may extend from the lowest levels of the spinal cord to the most recently acquired areas of the cerebral cortex.

It is important to emphasize that the perception or experience of pain is not necessary for the operation of the drive of pain. Animals with transverse sections of the brain stem at different levels will make such attempts as they are able to eliminate pain-fiber-stimulating agents, as will decerebrate preparations. And man under light anesthesia will do the same even though he " feels no pain." Undoubtedly, however, the perception of pain in intact humans, and presumably in other animals, may be regarded as contributing to the total motivation resulting from the stimulation of pain receptors. Whether or not the emotional response contributes is a question that we shall discuss later.

The Drive of Sexual Excitement. Much has been written in the last half century about the gonadal hormones and the " sex drive." In recent years, however, the increased amount of research on sexual behavior in man and other mammals has indicated that there is no simple one-to-one relationship between level of any gonadal hormone and amount of sexual activity. Most investigators agree, however, that sexual excitement or arousal has certain definite signs in mammals and that it is followed by augmented overt sexual behavior. Most will agree, also, that many factors enter into the occurrence or lack of occurrence of sexual excitement. In the next chapter you shall find that we regard sexual excitement as one of the primary forms of emotional behavior. Here we shall regard the organismic state associated with such arousal as the drive of sexual excitement, much as we have suggested previously that pain serves as a drive.

Sexual excitement in male and female mammals has some common and some different characteristics. Common throughout all species investigated is (1) tumescence of erectile tissues in the genitalia and other portions of the body, in particular, the nipples of the breasts; (2) a tendency to further the tactual or other stimulation that has elicited the tumescence; (3) first decreased, then increased muscular activity as stimulation is continued; (4) semiconvulsive muscular activities (orgasm) ; followed by (5) muscular relaxation. The behavior described in (4) and (5) seems to occur less consistently in females than in males, but may occur in both sexes of all species that have been investigated.

Many differences between males and females also are demonstrable: (1) males are more often aggressors; (2) males engage in more self-stimulatory behavior of all sorts; (3) single orgasms are typical for males, atypical for females; (4) females are more often passive, males more often active in reproductive behavior; (5) when receptive, females engage in sexual behavior more frequently than do males; (6) in infraprimate mammals, females usually will respond only during estrus whereas males of most species are responsive to sexual stimulations over longer periods of time.

The foregoing comments by no means exhaust the similarities or differences in sexual behavior among males and females of either infraprimate or primate and human species. They do serve to accentuate some of the similarities and differences. Concerning sexual excitement in general, tumescence of genital tissues seems to be its initial manifestation. Since it may be produced after total section of the spinal cord, it is essentially of reflex origin; and it is elicited by mechanical stimulation of some but not all tactile receptors. There is evidence that in some infrahuman mammals, certain olfactory or visual stimuli alone are able to elicit it; and certainly after learning has occurred, many kinds of stimuli may release it, especially in man.

Since tumescence may occur in young organisms it obviously does not depend on high blood levels of gonadal hormones. High androgen levels increase the frequency of its occurrence in both sexes, however; and in some infraprimate female mammals it rarely if ever occurs except when estrogen (and sometimes also, progesterone) level is high. Castration of young mammals markedly reduces it; but if castration occurs after puberty, the results vary with species, sex, and individual differences. In humans and male anthropoid apes, sexual activity may even be increased by such an operation, although it often is decreased in human males. In infraprimate males there usually is some decrease and, in females of some species, sexual behavior totally disappears after castration. It may be concluded that although levels of gonadal hormones are not always necessary for sexual excitement they facilitate its occurrence. The mechanism of this facilitation is not known, although some investigators believe that both male and female gonadal hormones act directly upon the hypothalamus.

The influence of gonadal hormones is most clear in the estrus behavior of most infraprimate females, a period of physiological and behavioral change associated with the reproductive cycle (cf. Chap. 15) and usually with ovulation. At such times, the animal becomes more active, her body odors serve as powerful releasers for male sexual arousal, and she becomes more receptive to males and other females and readily assumes the reproductive posture typical of her species. During her periods of estrus, but only then, she will engage in extensive sexual activity. Such behavior is known to depend on high levels of estrogen and, in some species, progesterone. After castration it disappears, but may be reinstated by hormone injections appropriate to the species.

Further evidence of the significance of gonadal hormones comes from experiments in which female animals have been injected with androgen. Although results are not entirely consistent, they most often include increased aggression and the assumption of male postures in sexual activity, in both normal and castrated females. Injections of estrogen in male animals have less specific effects but may facilitate the appearance of female sexual behavior. Gonadal hormones thus contribute to sexual excitement and influence in part the nature of sexual behavior, and the higher and more continuously maintained androgen level in males probably accounts for much of their greater aggression and total sexual activity.

One male characteristic that has not had the attention it deserves is the waxing and waning of the drive of sexual excitement in relation to sexual activity. Since male mammals typically discharge a small amount of semen at the height of sexual activity and, after one or several such ejaculations (depending on species and individual differences), do not again become tumescent or otherwise sexually active for some time, it has been believed by some investigators that the accumulation of seminal fluid itself contributes in some manner to sexual excitement in the male. Future research must determine the truth or falsity of the assumption.

In conclusion, it should be stated that the mechanism by which the drive of sexual excitement induces reproductive behavior is not known. It is certain that the drive is not simple, and probably we should speak of several drives which may differ in males and females and may operate in different manners. Moreover, the many other factors which influence sexual excitement must be taken into account. For example, some that are known to reduce it are fatigue, anxiety or fear, hypothyroidism and other endocrine deficiencies, and dietary deficiencies. Facilitating states are summed up rather neatly by one author who says that the best aphrodisiacs are good health, plenty of sleep and rest, and a full stomach.

Another way to approach the problem is in terms of the autonomic nervous system which is extensively involved in sexual behavior. We shall have more to say about that involvement in the next chapter. Here we wish merely to point out that tumescence is a parasympathetic reflex and excessive sympathetic activation in the sacral region prevents its occurrence. Thus, agents which selectively activate these two branches of the ANS will selectively facilitate or inhibit this major characteristic of initial sexual excitement.

It is not surprising, then, that research finds the hypothalamus necessary for integrated sexual behavior and suggests that it is here that the gonadal hormones have their effects. Other experiments have shown, however, that cortical lesions may selectively decrease [4] or increase [5] sexual activities and that male mammals are more affected than females by such lesions. Males also are more affected by the experimental destruction of olfactory, visual, tactual, or other senses; and some investigators now believe that for male mammals, in particular, sexual arousal and reproductive activity depend on the sum total of activation of the CNS resulting from gonadal hormones and appropriate sensory stimulation.

One other factor deserves comment here — the fact that once sexual excitement has occurred organisms attempt to further it, up to a certain point. In man, it is recognized as a form " pleasure." In Chapter 21 we shall discuss initial sexual excitement as a unique form of pleasure. In a later section of this chapter, we shall comment on pleasure in general as a drive.

Other Drives in Reproductive Behavior. As Table 20-1 indicates, care of offspring is just as necessary for species continuation as is sexual behavior, and the behavior having to do with such care is often attributed to " maternal " or " paternal " drives or " instincts." Since man seems to show little or no unlearned behavior in caring for his progeny, we shall give the problem scant attention. A word is in order, however, concerning such behavior in lower animal forms.

Some activities such as migration, the selection of breeding grounds, and preparation of a nest or burrow are known to be attributable to altered pituitary and gonadal hormone secretions and have been varied experimentally. Those that occur after parturition, or after egg laying and hatching, have been most studied in relation to female physiology. All such behavior shows wide species variation and is the subject of much research in comparative psychology and ethology.

Maternal behavior has been studied most thoroughly perhaps in the laboratory white rat. It involves a complicated pattern of nest building, parturitional postures, biting the umbilical cords, eating the placenta, cleaning the young, suckling them, and retrieving them if they stray from the nest. Such behavior typically occurs when levels of prolactin are high and parts of it may be induced in nonparturitional females or males when injected with this hormone. Since similar results have been obtained in other mammals, it might be said that prolactin serves as the drive for maternal behavior. Unfortunately, there is contrary evidence. Some forms of maternal behavior may be induced in nonparturitional females and in males by a number of means and after hypophysectomy. Obviously, we know little about the drive or drives that induce care of the young — hence the question mark in Table 20-1.

Other Possible Drives. We now have exhausted the classification shown in Table 20-1 but something remains to be said. In this section, we shall consider what makes dogs or cats explore new environments, what causes monkeys and human children to manipulate those portions of their environments that can be manipulated, and what accounts for play. Are there specific drives that induce these different forms of behavior, or can all be accounted for otherwise?

Most psychologists speak in some fashion of a " drive for exploration " because the fact has been established in many laboratories that mammals, such as the rat, not only typically will explore a new environment but may do so before reacting to other drives. Some authors speak instead of a " curiosity drive," especially if they are thinking of young children or monkeys. Some speak of a " drive to manipulate," or of a " drive to know," or a " drive to perceive "; and many speak of a " drive for play," because of its frequent occurrence in the young of most mammalian species. And, finally, many speak of a " drive for activity " which they may or may not relate to others mentioned here. At least one author puts them all together and claims that " capacity is its own motivation." [6] Or, to put it differently, there is a drive for each activity of which an organism is capable.

This last point of view is a tempting hypothesis but the evidence does not entirely support it. We know, for example, that many activities of which an organism is capable are induced solely by external stimulation, and, thus, many reflexes would be so classified. We have seen also, in *Machina speculatrix,* that persisting behavior may depend solely on structure (capacity) and external stimulation. So we see that the phrase " capacity is its own motivation " has little meaning. All persisting activity does not demand an underlying motive. For example, when the eyes of a human infant of three months of age focus on and pursue a moving light, we do not have to infer a " drive to perceive " or a " drive to know." Nor do we have to postulate a sensitizer. This is an activity of which the average three-month old child is capable and, in the absence of other prepotent stimuli, one which is elicited by a moving light.

One thing is fairly certain. Much spontaneous activity does occur in healthy growing mammalian organisms and probably in most others whose skeletal movements are effected by the specialized tissues we know as striate muscle. Although such activity often is attributable to other drives we already have discussed, it seems probable that it occurs also in their absence. We may speak then of an *activity-*

producing drive and search for it in the metabolites of growing muscle. Such activity in a new environment could easily be called exploration or curiosity, and in organisms that have the capacity for manipulation it might be so styled. Moreover, it might be called play.

If any such activities were in any way rewarding to the individual organism, they would not only be repeated but would become conditioned to specific external or internal stimuli or both. If such activities were nonrewarding or pain-provoking, however, they would disappear. This, it seems, is what we actually find. Not all animals who can do so actually explore, play, manipulate, or show curiosity. Some retreat from every new situation. For the present, then, we shall assume a drive underlying spontaneous activity in many growing organisms, but shall speak of play, manipulation, and exploration as particular and permissive manifestations of this general activity. Its most obvious counterpart in you, as sophisticated adults, is your tendency to stretch your muscles upon awakening, or your occasional desire to exercise just for the sake of exercise. Perhaps the most healthy of you may even experience what has been called "the joy of sheer movement." One psychologist refers to it as "functional pleasure." We shall refer to it again in Chapter 21.

Is There a Pleasure Drive? Pleasure commonly is regarded as an emotion, and the urge to continue pleasure-producing activities is strong in all of us and probably in other animals as well. We have indicated that pain and sexual excitement are emotional responses and also contribute drives. If you continue in your study of psychology, you soon will hear much about anxiety as a drive, yet you will discover in the next chapter that we treat anxiety as an emotional response.

Such statements about emotion and drive will be less confusing if you remember what we emphasized for pain and for sexual excitement: (1) that the effect of pain-receptor activation is not unitary, but typically involves perception, emotional response, and drive; (2) that initial sexual excitement, as a response, may set up new conditions in the body that induce other behavior.

It might be argued that all drives produce emotional responses and all emotional responses set up new motives, but we shall not pursue the point here. The first part of the argument is mentioned in the next chapter. The latter part must rest upon future research concerning the nature of emotional responses and their stimulus properties. So the answer to the question we have raised about a "pleasure drive" also must await a more detailed description of pleasure as a response.

We have raised the question only because it seems to be an important one that often is omitted from introductory treatments of motivation. And since we have raised it, we make a few predictions: (1) pleasure, as perceived by man, depends upon several different patterns of nervous discharge which induce different response patterns; (2) either the antecedent or consequent patterns of such nervous discharge, or both, may serve as drives in man and other animals. Moreover, similar statements may be made concerning " displeasure " or " distress." When such predictions have been tested we shall know a lot more about motivation than we do today.

Is Muscular Tension a Drive? In conclusion, let us state more explicitly that most drives have in common an arousal of greater tension in skeletal muscles and greater overt activity. This is true even though rather specific reflex adjustive acts may be associated with them. The human or other newborn organism does not know whether he is hungry, thirsty, cold, or has the colic; and his response to food deficit, water deficit, thermal deficit, or internal pain-receptor activation is much the same. He awakens, his muscles tense, he becomes increasingly more active, and sooner or later crying is added to the behavior pattern — one which is presumed to depend upon greater and greater activation of his nervous system. As the drive is reduced by appropriate measures, the crying and gross activity first disappear, then gradually the underlying tension decreases and he returns to a state of sleep or at least relative quiescence.

As he becomes older and comes to recognize specific drives and to make appropriate adjustments to them, the crying and undirected overt activity appear less seldom but the increased muscular tension still occurs. Since it is a response and sets up a new pattern of stimulation within the body, and since it is present during the conditioning of specific adjustive behavior, the sensory pattern arising from muscular tension comes to serve as a conditioned stimulus for a wide variety of behavior.

After such conditioning, if a new situation induces increased tension but no specific act, then other acts will occur that in the past have served to reduce tension and other drives. Thus, the young animal or child who is " distressed " by a new situation but does not know how to adjust to it, will typically do something else that has brought him " pleasure " in the past. He may eat just after eating, drink just after drinking, or masturbate.

It has become fairly common in psychology to speak of tension as a drive. Perhaps excessive muscular tension does have special drive properties and this certainly is true when it becomes so extreme as to produce muscular pain. To us, however, it seems more appropriate to look for the drives underlying tension, and to attribute tension-produced behavior not to a " tension drive " but to conditioning associated with muscular tension. For this reason we do not speak of tension as a primary physiological drive; but, of course, it may serve as a secondary or learned motive. Some emotional responses likewise may be regarded as the bases for secondary motives, while some — as has been suggested — may be considered as primary drives. Let us turn, then, to a consideration of emotional behavior and its significance in physiological psychology.

Suggested Readings

1. McClelland, David C., *Studies in Motivation*. New York: Appleton-Century-Crofts, Inc., 1955.
2. Morgan, Clifford T., and Stellar, Eliot, *Physiological Psychology* (Chaps. 18, 19, and 20). New York: McGraw-Hill, 1950.
3. Young, Paul Thomas, *Motivation of Behavior*. New York: Wiley, 1936.
4. Tinbergen, N., *The Study of Instinct*. Oxford: The Clarendon Press, 1951.

21 Emotional Behavior

Introduction. Emotion is a peculiar word. Almost everyone thinks he understands what it means, until he attempts to define it. Then practically no one claims to understand it. Scientists who investigate it disagree. Philosophers, novelists, and others who write about it disagree. But, in the meantime, we all go about our individual ways, sometimes enjoying our "emotions," sometimes bemoaning them.

Emotion is many things for different people. For some it is a mental experience; for some it is a way of acting; for some it is series of events occurring within our bodies; while for some it is a form of motivation. Many regard most emotion as "bad"; while others regard much emotion as "good." The Greeks wrote about emotion in much the same terms we use today, and sometimes it seems we have not learned very much about this subject during the last 20 to 25 centuries. Most of us still consider emotion important in life, however, regardless of how we define it or how well we think we understand it. But it is considered important for various reasons.

Those who consider most emotion as bad would hold that it should be understood so that we may better be able to minimize the disruptions in our lives that are attributed to it. They might speak of the nonutilitarian significance of emotion, cite evidence of the irrational judgments reached during fear or rage as compared with the rational judgments of unemotional contemplation, and emphasize the value of emotional control. We certainly should have to agree with them that sometimes emotion may do us harm; and, therefore, sometimes we should attempt to control it.

Those who consider much emotion as good would hold it important in enriching our lives, in removing the apathy of dullness. They might emphasize the value of emotion in learning, the value of anxiety in the construction of the codes of ethics and laws that make civilization possible, the impetus it affords to all thought and action. Here, too, we should have to agree in part. We all consider the unemotional person as either dull, or "cold-blooded" and calculating. We know that we remember well those situations which caused strong emotion in us. We know that anxieties for the future lead us to plan our lives;

and we know that excitement, fear, or rage gives us strength for acts which ordinarily we would not be able to accomplish.

Recently another reason for considering emotion as important has pressed itself on us. It is the growing realization that many of our diseases — whether called " mental " or " physical " — are intimately related to emotion. It has long been common to regard the **neuroses** and **psychoses** as associated with preceding extreme emotion. More recently we have discovered that a host of **psychosomatic disorders** — for example, asthma, ulcers, high blood pressure, and various skin ailments — also are associated with emotion. All in all, regardless of whether we consider most emotion as good or bad, we cannot escape the conclusion that emotional behavior is important in everyday life.

We shall not consider in this chapter many of the words man has used to describe his feelings or the more intense and sometimes more definite states he calls emotional; nor shall we review in detail the diverse psychological points of view. Others [1] have done this work more thoroughly than would be possible in a text of this nature and unless it is thoroughly done the end result is of little value. Furthermore, we assume that each of you has had some introduction to the topic of " emotion." Instead, we shall present here a specific point of view. At present it is largely theoretical in nature and, although parts of it are supported by considerable evidence, other parts need much experimental investigation. It might be referred to as " the visceral action " point of view.[2] Let us trace its development briefly and compare it with related points of view before stating it explicitly.

Emotion as Visceral Action. The point of view that action of the internal or visceral parts of our bodies is the salient feature of emotion, is both old and new. It is old in the sense that part of it was well described by Plato and Aristotle. It is new in the sense that much of it has been ignored or forgotten until recently, and in that the whole story has not yet been told.

Plato thought of emotion in terms of pleasure and pain and, with the early cynics, for him there could be no pleasure without prior pain. Aristotle gave us our first extensive classification of emotion and he used many terms which have followed us to this day. He emphasized the striving for pleasure and the attempted aversion of pain; thus, he might be referred to as the first dynamic psychologist. But he, Plato, and most of the early Greek writers emphasized that emotion primarily is an affair of the heart, the bowels, or the womb.

Such a theme runs through most descriptions of emotion (or passion, as it then was called) until the 18th century. By this time man had become convinced that the brain was important. Soon the brain came to be regarded as the " seat " of the passions (which by now usually were called emotions) and the subservient body was largely forgotten. Of course, the new breed of physicians never forgot it entirely but they were few and far between in a philosophical age. Gradually another breed appeared, who might be called " psychophysiologists "; and gradually we have witnessed a reinstatement of the importance of the affairs of the body in emotion. In short, there is a growing tendency to think about emotion in terms of patterns of psychophysiological response, particularly with respect to the functions mediated by the autonomic nervous system.

For some contemporary writers, emotion is a behavior pattern that occurs in the viscera and other bodily parts; but any emotional experience that occurs in the cortex is secondary. For others, emotion is a cortical event which depends upon response patterns that have occurred elsewhere — in the viscera, in the skeletal muscles, in the hypothalamus, or in some combination of these three. For some of both groups, the bodily pattern must be preceded by a psychological event if it, or subsequent mental events, are to be called emotion.

There are two entirely different points of view represented here, of course, and each is qualified. Let us restate them more specifically.

Class I. Emotion is a noncortical bodily event; consciousness of emotion is a mental event which depends upon the prior bodily event.

Class II. Emotion is a mental event which depends in part upon other bodily events.

Although these two statements seem to be similar they actually are quite different. For example, substitute the term " motion " for " emotion " and read them again. If we were to accept this second definition of " motion " then it would follow that we could know little about motion in a newborn human infant or in an animal other than man. Yet statement II is precisely the way emotion is defined by many people (both scientists and laymen) ; although often they find it easy to infer emotion in infants and subhuman animals from what they then call " emotional expression." If such " expression " occurs in a decorticate animal (who, presumably, cannot really be experiencing emotion) it then is said to indicate " sham emotion." Such circumlocutions serve only to muddle further an important area of behavior. They are unnecessary if we employ definition I.

Let us now consider three subclasses of these two points of view:

Ia. Emotion is vasomotor disturbance. (This was the view of Lange,[3] whose name often is erroneously combined with that of James.)

IIa. Emotion is a mental event. It is the feeling of changes occurring in the viscera and the skeletal muscles, which changes have been initiated directly by perception of an appropriate stimulus. (This is the view of James,[4] who referred to "perception of the exciting fact.")

IIb. Emotion is a mental event. It depends upon changes in the hypothalamus which have been initiated by an appropriate stimulus. (This is the "thalamic theory" of Cannon and Bard.[5])

These last two points of view have dominated psychology throughout its recent development, but both leave much to be desired. Perhaps most to be regretted is the fact that both claim that some bodily events (be they visceral or skeletal as in IIa, or hypothalamic as in IIb) do not produce emotion; but neither specifies the nature of the appropriate stimuli for those changes that will produce emotion. Some writers attempt to resolve this problem by claiming that emotion occurs only when *learned* motives are involved; thus, suffocation or hunger would not be emotion or produce emotion, unless the threat to life was perceived and reacted to. Although such a view is commonly held and may appeal to you since you are used to it, we do not find it convincing. It would have to deny emotions to newborn organisms; and it would have to hold that identical patterns of bodily events sometimes are emotional, but other times are nonemotional.

Such problems are resolved, however, if one adopts a class I definition and rigorously defines the bodily event that shall be called emotion. Let us see how this works out in some statements concerning emotion as visceral action.

(1) Altered activity of the autonomic nervous system and the tissues or organs that it innervates is the sole factor common to the majority of stated or implied definitions of emotion or emotional behavior in man or other animals, whether described by scientist or layman.

The logical reduction of such a proposition becomes,

(2) Altered activity of the autonomic nervous system and the tissues or organs that it innervates is a sufficient and necessary criterion of emotion or emotional behavior.

This appears to be a straightforward statement except for the term "altered activity." Presumably, each such alteration is measurable, and one might modify the definition by stating the units of change required to satisfy the designation, "emotion." Such an approach to the problem would be comparable to defining the number of inches a child must grow before it may be considered "tall," or the amount of air that must be forced into a balloon before it can be considered " big." Let us utilize, instead, a continuum such as length or volume.

Let us assume the bottom limit of our continuum for organismic emotional behavior as " death," a state in which there is no activity or reactivity of the ANS. A next step on the continuum might be that demonstrable in deep anesthesia, when the ANS is active but relatively unreactive to new stimuli.

Now let us delimit the term " emotion " as a noun indicative of a state of internal (visceral) motion or action, then add permissive statements concerning skeletal muscle activity and mental activity, and then reword our proposition.

(3) Emotion is activity and reactivity of the tissues and organs innervated by the autonomic nervous system. It may involve, but does not necessarily involve, skeletal muscular response or mental activity.

In living organisms equipped with autonomic nervous systems, emotional behavior would thus be continuous and the task of investigation would be to measure the degree of behavior in different situations in terms of (a) activity and (b) reactivity. Techniques would have to be devised for valid measurements of many of the organs involved; for some they are available, for others considerable work already has been done.

Extensive research has been reported, for example, on measurements of electrical conductance of the skin and of the **galvanic skin response (GSR)**; the first, a rough measure of level of activity; and the second, a fairly precise measure of reactivity of sweat glands, which are known to be innervated by fibers of the sympathetic branch of the ANS. By our definition, both measures would be expressions of emotional behavior and any change in either measure would indicate a change in emotional behavior.

Work with these measures, however, forces two additional considerations to our attention. In the first place, the autonomic fibers that innervate the sweat glands are cholinergic in nature and are not reactive to adrenalin as are the adrenergic fibers. We find, then, a pattern of increased SNS activity following injection of adrenalin, but no marked change in sweating. Furthermore, different patterns of sweating are well known, and many researchers have demonstrated change in conductance level and GSR without finding changes in other sympathetically innervated organs. These considerations may be stated as two corollaries:

(A) Change in emotional behavior may be detectable in one autonomically innervated tissue or organ and be undetectable in others.

(B) Change in emotional behavior may be detectable in a part of one autonomically innervated tissue or organ and be undetectable in other parts.

With these corrolaries in mind, we may state an extension of proposition (3):

(4) Change in emotional behavior is altered activity or reactivity in a part of one, or more, tissue or organ innervated by the autonomic nervous system.

The investigator who accepts propositions (3) and (4) must devise techniques for simultaneous measurement of level of activity and degree of reactivity of many autonomically innervated functions before he can describe, predict, and control emotional patterns. Concerning some such functions there already exists much suggestive evidence, some of it quite specific, which may be employed in the formulation of initial hypotheses. Within our own observations of emotional behavior in ourselves and others, and within the observations of many others as recorded in the history and literature of all mankind, we have at the moment certain insights into differential physiological patterning of which we have made little use. At the very least, they furnish us certain tentative hypotheses around which to design more decisive laboratory experiments. Actually, some fairly definite steps have been taken in experimentation on physiological patterning and they will be referred to later. Let us consider first this personal information which is available to us all and consider some possible dimensions or axes of emotion.

SOME POSSIBLE DIMENSIONS OF EMOTION

Undifferentiated Excitement. Psychologists have long regarded excitement as one of the axes of emotion. Many authors speak of an undifferentiated excitement to distinguish it from excitement which is characterized by " fear," by " anger," or by " pleasure." We would conceive of undifferential excitement as a response to any undifferentiated situation, with increased ANS activity in which the sympathetic branch is markedly dominant. A close analogue to it would be the reaction following injection of adrenalin, but at least one major difference exists. Typical excitement as observed and recorded involves rather marked perspiration from the palm of the hand and other emotional sweating areas, whereas this reaction does not occur following the injection of adrenalin.

The Axis of Fear. Closely allied to undifferentiated excitement would be a number of different but related patterns for which we have a rich terminology. The word " terror " presumably would mark the extreme of one axis and such words as " apprehension " and " anxiety " would characterize a less intense degree of involvement. For many years it has been known that the sympathetic branch of the ANS is extensively involved in fear reactions and it seems possible that fear, as we now know it, involves one of the most clear-cut physiological patterns that will be found. We even use the term, " cold feet," as descriptive of cowardice, and cold hands and paleness are also typical concomitants. Only slightly less obvious and less well recognized are the wide eyes and dry mouth of the fear reaction and it has been shown that the " lump-in-the-stomach " reaction is related to inhibition of peristalsis. Fear, then, is held to be characterized by practically a pure pattern of sympathetic dominance and may differ mainly from undifferentiated excitement only in terms of its psychological setting. It is believed to represent a reaction to anticipated frustration and, because of its anticipatory characteristics, to be dependent upon learning by the organism. By this analysis, of course, we would speak of no unlearned fears. The original pattern would be one of disequilibration to noxious stimuli, and the fear pattern would be one which becomes anticipatory as a result of one or more past experiences.

The Axis of Pain. Closely related to the fear pattern would be that of pain, however there seems to be sufficient evidence to hypothesize that there will be some differences, particularly in the pattern of sweating, weeping, and perhaps also heart action. It is probable that the essential characteristic of pain is not a particular pattern of autonomic activity, but a particular pattern of sensory activity; but we would still speak of an emotional axis of pain, with " agony " at one extreme and " mild discomfort " and " distension " at the other. Among the senses, pain receptors are prepotent in eliciting SNS activity.

Exercise. Although exercise is a well-known physiological pattern, it is seldom thought of as an emotion. By our usage of the term, however, it falls within this classification since it involves altered activity of the autonomic nervous system. The change in heart and respiration rate and the panting and perspiration associated with the dissipation of heat are well-known aspects of strenuous activity of skeletal muscles. Undoubtedly, portions of the sympathetic branch are actively involved, but we do not find the peripheral state of vasoconstriction which is characteristic of the pattern of fear. Instead, with the pattern of heat dissipation we find marked vasodilatation in the peripheral bood vessels. Although the total autonomic pattern in exercise has never been studied as such, we can easily infer some of the other changes. There would seem to be a marked decrease in gastro-intestinal peristalsis for we find, for example, in the person who is exercising strenuously, a tendency for that characteristic "lump-in-the-stomach," especially if any food has been ingested just prior to exercise. The size of the pupil apparently has not been reported accurately, but there is some suggestion that dilatation occurs; and, although specific studies have not been made of salivary output, the mouth frequently becomes dry with strenuous exercise. Whether or not this is a result of increased evaporation due to mouth-breathing, or whether it is due to increased sympathetic stimulation of the salivary glands, is a matter for future research. It seems probable, however, that the physiological pattern of exercise is characterized by rather extensive activity of the sympathetic branch of the autonomic nervous system with a superimposed pattern of heat dissipation.

The Axis of Anger. Perhaps it is not surprising that the physiological pattern of anger, in so far as we can infer it from casual reports in the literature, is similar to that for exercise. Our own observations, coupled with those of writers for centuries, hold anger to be a "warm" emotion and not characterized by "cold hands" or "cold feet." Possible exceptions may be found to these statements in the expressions, "cold anger," or "cold rage," or in such descriptions as, "He was pale with rage." These possible exceptions may be explained, it is believed. The cold anger, in the sense of a calculated response, would be characteristically intellectual in nature and need not be emotional. The cold anger characterized by cold peripheral skin areas, which also would be pale and relatively bloodless, might possibly be explained as an atypical, learned, behavior pattern.

The typical physiological pattern of anger remains to be determined in the laboratory. It probably will be very difficult to artificially arrange true anger situations, but it may be predicted that if and when they are so arranged, the pattern will be something as follows. It will involve marked activity of the sympathetic branch of the ANS but there also will be strong reflexive parasympathetic components; particularly, increased salivary output, increased secretion of hydrochloric acid, increased peristalsis, and peripheral vasodilation.

Disappointment. Let us pass now to the consideration of another axis which in its initiation may be rather closely related to the pattern of anger and annoyance. In adults, both occur apparently in response to specific types of stimuli. In anger, the adult individual believes that he can do something about the matter or that he wants to do something about the matter; in disappointment, however, he believes that he cannot do anything about the matter. He accepts the situation and does not show the typical " emergency response." It is believed that the axis represented by disappointment, sorrow, grief, etc., is not a simple one. It is probably very complex and may consist of several different kinds of autonomic nervous system patterns.

When we use such terms as sorrow or grief, we typically imply a previous period of excitement. The final pattern is believed to be one of overcompensation on the part of the parasympathetic system. Here also, however, we have much to learn about the true physiological patterns involved. Even though the lachrymal or tear glands are innervated by the parasympathetic system, it is to be doubted whether they indicate an extensive involvement of that system. Perhaps we should regard crying in sorrow or grief as a reflexive pattern which is super-imposed on an actual depression of both branches of the autonomic nervous system.

The Axis of Sexual Excitement. A well-known emotional pattern is one which we might call sexual excitement. It is clear that in the early phases of this type of reaction, at least, the sacral portion of the parasympathetic system is dominant. The erectile tissues of the genitalia are known to be innervated by this group of nerves; and furthermore, in the presence of marked sympathetic stimulation erection does not occur. Although there is some indication that increase in heart rate occurs in sexual activity, the pattern is by no means clear. There is further indication that the act of ejaculation, and possibly orgasm, involves reflex activity of the sympathetic system. Sexual excitement, thus, seems to be very complex with at least an initial sacral phase, followed by a short period of sympathetic dominance, after which there seems to be a secondary phase of parasympathetic dominance associated with overcompensation by this latter system. This last pattern in sexual behavior will be more clear after we have considered " relief."

Initial sexual excitement undoubtedly represents one aspect of " pleasure " in human beings. This feeling or experience presumably is associated with mild tactual stimulation in general, but in particular is elicited by stimulation of those portions of the skin which are called the **erogenous zones.**

Relief. A second form of pleasure is believed to be associated primarily with the satisfaction of needs vital to the individual organism, i.e., with relief from basic physiological drives; thus, it would be conceived of as " basic pleasure." As experienced by human beings, it is believed to involve a distinct physiological pattern associated with a reduction in sympathetically innervated responses and possibly with mild overcompensatory activity on the part of the parasympathetic nervous system. You know that almost any type of stimulus induces increased sympathetic action. After the stimulus to sympathetic function has been removed, the parasympathetic system may overcompensate slightly before it, itself, returns to its prestimulation level of activity. This, it is believed, is the pattern known as relief, satisfaction, or gratification. Once it has been experienced and the anticipation of its future experience has been attached to symbols in the external environment, we may find patterns of anticipatory pleasure which may be called pleasant excitement or even elation. Before anticipatory pleasure could be demonstrated to a given situation, however, the organism must have experienced relief in a similar situation (i.e., removal of a noxious stimulus and consequent reduction in sympathetic activity with overcompensatory parasympathetic activity).

Other Possible Forms of Pleasure. One might argue, as did Plato and the Greek cynics, that there is no pleasure without prior pain. From such an argument one might conclude that relief from pain is the only true pleasure, and that all other pleasant experiences are conditioned upon it.

We already have pointed out, however, that the visceral response pattern in initial sexual excitement is different from that in relief. Since there seems no good reason for assuming that pain must precede it, and since there is good reason for assuming that both give rise in man to experience which is called "pleasure," we are forced to conclude that there are at least two forms of pleasure. The question arises — are there others? At the moment we cannot answer this question. We can only add that at least two other forms have been suggested which deserve experimental investigation.

One has been called "functional pleasure" and it is held to be important in the play of young children and other vertebrates. It refers to the kind of experience we have when we speak of the joy of "sheer movement." Sometimes it is employed in an attempt to explain the fact that certain auditory rhythms are so widely appreciated (presumably, via the motor behavior they elicit).

The other has sometimes been called "perceptual pleasure" and carries the implication that certain perceptual experiences (for example, certain odors or tastes, certain auditory rhythms, or certain visual forms such as a given shape of nest for a given species of bird) are sought after without apparent learning, while other perceptual experiences are averted. Only further research can determine whether we should retain these or other possible "pleasures" among the dimensions of emotional behavior.

Specific Reflexive Patterns. Undoubtedly there are many specific reflexive patterns involving portions of one or both branches of the ANS which should be included in any category of emotional behavior as we have defined it. Some of these are more frequently considered as emotion than others; for example, disgust and embarrassment. It is obvious that embarrassment involves a specific form of autonomic reflex. One of the characteristic signs in human beings is blushing, or vasodilatation, in the head and neck regions; and we would point out the fact that you now use this autonomic pattern as your real criterion for judging the presence or absence of embarrassment. The remainder of the pattern has not yet been thoroughly investigated, but this is one emotional response, at least, which should be capable of rather thorough investigation in psychological laboratories.

The same should hold true for the pattern of disgust or revulsion; and here we have one good example of laboratory research which gives strong support to the claim that the basis for any response which we describe as disgust lies in reflexive parasympathetic activity involving increased tone (sometimes to the point of reversed peristalsis) in the gastro-intestinal tract.[6] In many instances when the word disgust is used, it is obvious that there is little or no change in autonomic responses. Instead, the response is purely on an intellectual level, much as the " I'm happy to meet you " reaction, which may be entirely verbal and completely unemotional. Only if there is increased activity of one or more portions of the autonomic nervous system would we speak of increased emotion.

Another reflexive pattern which sometimes has been included in categories of emotion is that known as " startle." As we shall see later in this chapter, this reaction is one fundamental from which more extensive patterns of fear arise. The startle pattern is soon terminated in the absence of continuing stimulation, but regardless of its fleeting character we must regard it as a true emotional response. It involves a definite change in ANS activity.

One other definite reflexive pattern deserves some discussion. It has no name except " shudder," yet this term is hardly appropriate. It is the response which we give to the sound of a hard chalk on a blackboard, to almost any " grating " noise, and perhaps to sandpaper rubbed on the skin. Its pattern is known to involve pilo-erection and a drop in skin resistance. By our definition it is certainly an emotional response and it deserves more extensive investigation.

In addition to the reactions already mentioned, the autonomic nervous system is known to respond rather extensively in many other situations. Reactions to humorous situations or to tickle would be two examples, one predominantly intellectual in nature, the other predominantly physical. But the latter, as well as all ANS reactions to temperature, to internal infection, and to drugs and toxic conditions, are to be considered as changes in internal action — as changes in emotion. There probably are hundreds of such behavior patterns, but we do not know. Until they have been investigated in the laboratory we can only hypothesize, as we have done concerning some patterns which seem of most importance for psychology. Finally, we should say a word about patterns of reduced ANS activity.

One author already has called " sleep " a parasympathetic reflex. Whether or not it is entirely parasympathetic in nature, it certainly does represent a change in autonomic function and, by our definition, a change in emotion. Sleep and rest probably represent the greatest reduction in sympathetic activity that may be observed in the daily behavior of normal organisms. As such they serve as a fitting introduction to a discussion of depression.

Depression. At least one other form of change in emotional behavior deserves particular mention. It most typically is referred to as " depression " and is most often confused in both lay and psychological literature with patterns of grief, sorrow, and apathy. It is reserved here to describe a condition in which at least the sympathetic branch, and possibly both branches of the autonomic nervous system, are in a hyporeactive state.

There is some experimental evidence for this point of view. In so far as we know anything about the physiological pattern shown in extreme depression in man, it may be described as just this hyporeactive autonomic state. There often is reduced metabolism; and while there may or may not be reduced heart rate and blood pressure, at least changes in such functions are very much minimized.

THE REINSTATEMENT OF HOMEOSTASIS

During and following any kind of increased emotional activity, one finds adjustive mechanisms at work apparently directed toward the re-establishment of homeostasis or autonomic equilibrium. The primary mechanism by which this end is achieved is overt activity which eliminates or reduces the noxious stimuli that originally elicited the increased emotion. Secondary mechanisms include the behavior patterns known as rest and sleep. Less frequently recognized as adjustive mechanisms are the various reflex patterns which appear in skeletal musculature during any extreme form of increased emotion. They quite frequently are known as " motor discharge," but this would seem to be an unfortunate concept. The question rarely is asked, " What is being discharged? " But it usually is implied that there is some form of " emotional energy " or " emotional tension " which must be reduced.

If we accept the argument that " emotional tension " is really striated muscle tension, we might be led to the assumption that the " discharge " of this tension itself reduces emotion. Emotion defined as autonomic nervous system activity, however, does not necessarily include tension in skeletal muscles. Although quite frequently with increased sympathetic activity there is increased striated muscular tension, there is no known autonomic innervation of striated muscles. The effect would seem to be an indirect one, perhaps consequent upon stimulation of the prefrontal lobes of the cortex via hypothalamic projection fibers. We would regard increased muscular tension, then, as a concomitant of some increased emotion — not as emotion itself. It may be conceived of as part of an organismic " emergency reaction " which serves to prepare the individual for more effective fight or flight, or for specific reflex activity; and with appropriate stimulation any of these overt acts may follow it.

It is doubted, however, whether overt muscular patterns as such represent the same kind of adjustment in all increased emotional activity. That is, the " motor discharge " view is too simple. Overt patterns may be found in one form or another for most emotional change except the minor reflexive autonomic patterns, but they vary considerably. The writhing of agony and the restlessness and hyperactivity of elated or undifferentiated excitement are well known, as are the rapid retreat of fear and the attack of anger. In some animals, the latter two forms of behavior, at least, seem to be preceded by characteristic skeletal reflexes which appear to be organized at the thalamic level. Thus, the decorticate cat, but not the midbrain cat, will demonstrate organized patterns of (1) arching the back, growling, hissing, spitting, and baring the claws, or (2) cringing and mewing. So, too, will man continue to show involuntary reflexes in the face musculature after supra-thalamic lesions have destroyed voluntary control of facial expression. Many other overt reflexes which often occur with emotional change also seem to be organized at the thalamic level; such as sobbing or crying, and laughing or smiling.

Although some of these muscular movements themselves presumably set up other conditions in the body which counteract the originally stimulating situations or alter subsequent internal stimuli, many, such as the writhing of agony or the restlessness of elation, or various facial expressions, seem to have no purposive nature. The minor patterns may be, as Darwin believed, only vestiges of our evolutionary history; but, for the major patterns of muscular activity and the part they presumably play in the readjustment of the organism, there must be some explanation other than merely a " discharge " of muscular tension. If running around the block does serve to reduce ANS activity in anger, if the activity of writhing or clenching of fists or jaws does serve to reduce ANS activity in pain, there must be some explanation of it in terms of reduction of stimulation of autonomic activity. Two possible explanations may be suggested. One might be called " distraction," and it would imply that attention to the new activity serves to reduce the effect of the emotion-producing stimuli. A second may be called altered internal environment, and it would hold that extensive muscular activity itself produces a new biochemical state in the organism which inhibits SNS activity and facilitates PNS activity. There is little doubt that the first of these explanations is valid in some emotional situations. There is considerable evidence, also, for the second explanation, but since its mode of operation is not clear at present, we shall not discuss it further.

Not all intense emotion occurs, however, in situations where strenuous muscular activity is possible. Furthermore, other factors that may serve to redirect attention away from the stimuli which originally evoked the increased emotional pattern cannot always operate. If noxious stimuli are intense, they unremittingly receive our attention and suggested distraction, or increased muscular activity, have little effect. In such situations it is believed that another form of adjustive mechanism makes its appearance. It might be referred to as the " zone of excitement tolerance " of organisms and the end result is the familiar fainting response.

Fainting in fear or in other excitement is very common, and the pattern seems to occur most frequently and most rapidly in situations which usually do not normally induce extensive overt muscular activity. There are occasional reports in the literature where fainting has occurred during extreme anger, when the overt muscular expression of that anger has been inhibited. Fainting frequently follows the sudden reception of catastrophic news about which the individual can do nothing at the moment. Furthermore, it occurs occasionally during intense sexual excitement, especially if orgasm is inhibited or prevented. Here the term most typically used in the literature is " swoon."

The pattern of fainting may be viewed as a strong parasympathetic compensatory reflex. Except in orthostatic hypotension (low blood pressure on standing), or following direct carotid ulation, it seldom occurs in the absence of prior strong stimulation of the sympathetic system. The actual mechanism involves a marked slowing of the heart with peripheral and splanchnic vasodilatation, with a consequent reduction in blood pressure and a loss of blood from the cerebrum. It is a pattern which would appear to occur more easily and more frequently in some individuals than in others, and one that presumably involves a strongly reactive parasympathetic system. In man, at least, it is assumed to occur when the organismic tolerance for sympathetic stimulation has been reached. We have little insight at the moment as to the nature of the actual predisposing factors. It is obvious, however, that the pattern marks a temporary phase of parasympathetic dominance and that it quickly and effectively removes the originally exciting stimuli from awareness or consciousness. Following a return to consciousness the stimuli may still be present as, for example, in chronic pain, but the organism has at least had some respite.

Muscular and Mental Aspects of Emotion. In emphasizing emotion as visceral action, we have intentionally neglected other aspects of emotion; but you will remember that we did provide for them. We said that emotion may be accompanied by changes in striate muscle action and by changes in mental activity. We pointed out that increased tension in skeletal muscles typically accompanies some patterns of emotion but it also is reduced in others, such as relief. More specifically, there seem to be certain patterns of muscle tension associated with some emotional patterns; such as the flexion pattern in pain and fear, or the extension pattern in anger, or the different " bowed-back " flexion pattern in grief or depression. Still more specifically, there seem to be some characteristic patterns which involve the facial muscles, the vocal and respiratory muscles, and — in some animals — the claws. These so-called " expressions " of emotion have been much studied but, for man, there is little agreement about their consistency or their significance.

You will see in the next section that we regard some of these striate muscle patterns as indicators of emotion in human infants. Since adult man can learn to suppress them and still experience and demonstrate emotional behavior, we cannot regard them as essential criteria in human adult emotion. Some writers would hold that alterations in general muscle tension are an exception and deserve an equal place with ANS changes in a definition of emotion, but we believe that the autonomic system furnishes the salient features of emotion and that muscular tension or relaxation may be construed as a proprioceptive background which facilitates, inhibits, or otherwise modifies our emotional behavior and our overt activity.

With respect to mental activity, let us emphasize that we have no desire to minimize its importance in emotional behavior. We wish, merely, to get emotion per se " out of the mind." That we all are aware of and experience emotion is unquestioned. That we perceive external stimuli as potential satisfiers or annoyers and react to them emotionally is obvious. That we perceive certain visceral events (such as a stomach cramp or a fast-beating heart) and report them as " feelings " is also clear. That we vaguely perceive certain other visceral changes and report them as feelings of happiness, anger, disgust, embarrassment, or depression is our contention. But when one uses such words to report a change in " feeling " in the absence of real change in visceral functions, then we should have to say that no change in emotion is represented, no perception or feeling of change, and, therefore, that the reaction is ideational in nature. Many of our so-called " pleasures " or " displeasures " are solely mental reactions. We say we are " happy " about something, or " displeased " at something else; we say we " hate " to lose a football game, or that we " love " our country or our dog. Most such verbal reports are expressions of attitudes, of ideas. In the absence of altered visceral action, they are not reflections of changes of emotion. In the presence of altered visceral action, they represent a mental concomitant of emotion.

AN ONTOGENETIC APPROACH TO EMOTION

A number of years ago a psychologist claimed that there are three primary emotional behavior patterns to be found in newborn human infants.[7] He called them *x*, *y*, and *z*; but believed them related to fear, rage, and lust. According to him, pattern *x* (fear) is avertive behavior induced by loud noises or falling; *y* (rage) is a struggle reaction to interference with freedom of movement; and *z* (lust) is a tonic inhibition of behavior (with or without smiling, or penis erection in the male) elicited by mild stroking of the skin, especially around the genitalia.

A number of experiments have been reported which claim to negate this theory, and few writers now follow it. The fact remains that no one has proved it wrong. We shall take the position that it is more right than wrong; and that its greatest error is one of omission. It did not go far enough.

Let us consider these three infantile behavior patterns and ask the following questions: (1) Are these responses similar to emotional behavior patterns in adults? (2) Is the list complete, or are there other responses of which the human infant is capable that are similar to adult emotional responses?

Perhaps the response about which we may most readily agree is the one called rage. We know that both infants and adults are apt to struggle against something or someone when their physical movements are interfered with, and in so doing they are apt to become warm and flushed. Since, as adults, we are likely to admit of anger at such times, let us tentatively agree that the struggle response in the infant has something in common with adult anger.

So, too, may the *z* response be related, at least, to initial sexual excitement in adults which has been elicited by the sort of tactual stimulation we call the caress. In the adult and in the infant, the pattern of reduced general activity with tumescence of erectile tissues is quite similar.

With respect to the *x* pattern, which at first is one of startle, not fear, we also can see certain immediate similarities to the type of adult behavior we call surprise, or even startle. In fact, this is one of the few patterns of so-called emotion that has been extensively studied in both infants and adults[8]; and the authors of the most thorough study leave little doubt that our adult reaction to sudden loud sounds or other sudden intense stimulation represents only an adult modification of the infantile startle pattern.

Let us turn now to the second question. Are there other infantile response patterns which are similar to adult emotional behavior? We shall take the position that there are at least five.

One of these patterns is so similar to the startle pattern that they may be practically indistinguishable. This is particularly true if the stimulus (for example, an electric shock or quick jab of a needle applied to the foot) is sudden and of short duration. If the stimulation is less abrupt and persists for some time the response becomes varied and usually involves crying; but always there is exaggerated flexion of muscles of the stimulated part of the body. It seems reasonable to call this pattern of behavior an " exaggerated flexion response " to painful stimuli and to regard it as similar in infant and adult.

Less dramatic but none the less consistent is the gradual increase in general activity which occurs with altering bodily conditions that might be described as progressively more noxious, such as those associated with increasing hunger and thirst. If the frequency or magnitude of an infant's activity is measured minute by minute and plotted against elapsed time since feeding, we find there is a more or less steady increase. Finally, crying sets in and the " mass " activity usually continues until feeding. At that time, apart from sucking and other feeding movements, there is an abrupt decline in activity, and if feeding is continued as long as sucking movements occur the infant typically falls asleep, becomes very "sleepy," or at least is very quiet. In the latter case, there may be some gurgles or cooings, and smiling, all of which fond parents are likely to accept as signs of pleasure. Let us, for the present, merely say that prior to feeding there existed a general activity response which certainly involved increased excitation to skeletal muscles and other parts of the body, but that after feeding we find quiescence and repose. The first pattern has been called "undifferentiated excitement" and seems to be related to excitement in adults. The pattern of "post-stimulation quiescence" has long been referred to as one of satiety, contentment, or relief.

Of course the flexion response to painful stimuli and the excitement response to other noxious stimuli themselves have certain similarities, among both infants and adults, but their differences would seem to deserve emphasis. For the one, if flexion is successful in averting the noxious stimulus, the child soon learns to make avertive movements at signs of the approach or threat of such stimuli. For the other (i.e., for situations of persisting discomfort from external or internal sources), he gradually learns ways of reducing or eliminating the underlying noxious stimuli. He learns to say he is hungry; he learns to get a drink when he is thirsty; he learns to go to mother, or to others for help, and so on. Regardless of the nature of the response, if it is successful in removing or even greatly reducing the noxious stimulation we find the relief pattern. As we already have indicated, it probably is our first and most prevalent basis for what we call pleasure.

Another response pattern in the infant is elicited by certain forms of chemical stimuli, especially those that are tasted. In general, sweet substances will elicit maintained sucking activity; but any strong salt, bitter, or acid solution will be spat out with puckering of the lips and contraction of other facial muscles, and the mouth will be averted from further stimulation if possible. The same pattern may be seen if the HCl (or "sour") content of the stomach is high when vomiting occurs. It seems logical to assume that this "spitting-mouth aversion" pattern is closely related to adult behavior. That we often show the same pattern in a situation which we call "distasteful" or "disgusting" (a word of the same root as gustation) is significant. And, with respect to the added reaction of vomiting, we frequently hear such expressions as, "It was so disgusting I wanted to vomit." You already know that one experimenter has reported increased gastro-intestinal tone and even reversed peristalsis to be associated with " disgusting " stimuli. We don't know that this latter pattern is a part of the " spitting-mouth aversion " pattern in infants, but it seems safe to assume that there is some relationship between this infant response pattern and truly distasteful experiences in adults.

The significance of the eighth infant response pattern we shall discuss is not so clear as the others, nor is it so easily described. For want of a better term we shall call it the " exhaustion-whimpering " pattern. It refers to the kind of behavior an infant displays after a long period of mass activity which has not produced removal or marked reduction of the stimulus. The strenuous activity gradually subsides and we find atonic or flaccid muscles, and the former screaming, or rapid and loud crying, is soon describable as sobbing or whimpering. In the older child such crying may continue for long periods, and we may hear the comment, " He's crying as though his heart would break." For the disappointed adult the muscular activity may have been inhibited so that the " exhaustion " portion of the pattern is doubtful, but we still may find the flaccid muscles (usually after a period of tension and excitement), and often we find the weeping. Perhaps, then the infantile " exhaustion-whimpering " pattern has something in common with adult behavior in grief or despair.

These infantile patterns and their suggested emotional correlates are summarized in Table 21-1.

Table 21-1. Infantile Response Patterns and Their Possible Correlates among Adult Emotional Behavior

Infantile Pattern	UCS	Adult Emotional Axes
1. Startle	Sudden intense auditory or visual S	Surprise
2. Struggle	Interference with movement	Anger
3. Tonic quiescence-tumescence	Sustained, gentle tactual S	Initial sexual excitement
4. Exaggerated flexion	Sudden stimulation of pain fibers	Pain
5. General activity	Sustained noxious S	Excitement (or sustained pain)
6. Post-stimulation quiescence	Removal or marked reduction of noxious S	Relief (pleasure)
7. Spitting-mouth aversion	Noxious gustatory S	Distaste (disgust)
8. Exhaustion-whimpering	Persisting and unrelieved noxious S	Grief (despair)

You will note that the stimuli which initially are effective in eliciting these eight infantile responses are referred to in Table 21-1 as "unconditioned stimuli." The implication is, of course, that any of these patterns may become conditioned to other stimuli. Also implied in the previous discussion is that each of these overt behavior patterns has associated with it a particular pattern of visceral action. We should expect, then, that these eight visceral action patterns also would be conditionable and gradually would become elicitable by all sorts of originally ineffective stimuli.

As we grow and learn, we often greatly modify our overt response patterns, sometimes suppressing them almost entirely. We learn that it isn't manly to whimper or scream, and that it isn't proper to spit food at the dinner table, or to strike everyone who angers us. And this learning, with our so-called "voluntary muscles," is fairly easy. It is more difficult, however, to control our so-called "involuntary muscles" and the glands of the viscera. Furthermore, only rarely is social pressure brought to bear towards their control. Thus, it seems reasonable to assume that these eight visceral action patterns will continue to occur in most of us, whether elicited by unconditioned or by conditioned stimuli.

These visceral patterns, of course, are what we regard as emotional change. And we can find no good reason for demanding that the term "emotion" be reserved for visceral changes elicited only by conditioned stimuli or for mental changes that occur in such situations.

Closing Comments. Probably most of you, and many others, will go on for many years believing that running for exercise is not emotion, but that running from a bear is emotion. Perhaps you will even hold that a child's reaction to its first needle injection is not emotion, but only pain; while its subsequent reaction to the prospect of another needle injection (or to the doctor, or to anybody in white) is true emotion. You will not be alone in your beliefs. Many will join you. They will say, "One is pain, the other is the emotion of fear, and that's all there is to it." Before you revert to such opinions, however, and forget entirely the point of view that we have tried to present, let us ask you one question. How do you know that another person is embarrassed? After some social error he may attempt to continue his conversation or other activity as though nothing had happened and he may even deny that he is embarrassed. But a visceral action pattern has occurred, a part of which is rather specific and detectable by you. He has blushed! And you have no hesitation in making your judgment. He is embarrassed!

Someday the various visceral action patterns of which man is capable will be known and methods will be available for their detection. Then, and not until then, can we hope to understand emotion. In the meantime we urge you to " keep an open mind " on the subject. We can only hope that this chapter will stimulate you to think about this interesting and important aspect of behavior.

Suggested Readings

1. Dunbar, Helen Flanders, *Emotions and Bodily Changes,* 4th Ed. New York: Columbia University Press, 1954.
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22 *Psychosomatic Problems*

The " Mind-Body Problem." We frequently hear today the terms **psychosomatic disorder** and **psychosomatic medicine.** It is unfortunate perhaps that such a term as " psychosomatic " ever was coined, reviving as it does the " mind-body problem," and the implication that the mind as a separate entity affects the body. But the so-called " mind-body problem " hardly exists for psychology today. In this text we have held that the term " mind " has little meaning for contemporary psychology apart from the activities of one of the body organs, the cerebral cortex.

We have seen that through this remarkable bit of cortical tissue, when it is adequately nourished and not interfered with by other bodily functions, we are able to do some rather astonishing things. We become aware of our external environment and aware of some aspects of our bodily states and functions; we remember and learn, and thus are able to control many of our body functions and many aspects of our environment; we develop concepts for both concrete objects and abstract symbols; and, finally, we utilize these concepts and other memories in various manners which we call thought, imagination, ideation, or reminiscence. All of these activities, of course, are called " mental." If we are aware of them as they occur they are called " conscious." If we are unaware of them, they are called " unconscious." But so far as we know, all essentially are activities of the cerebral cortex.

If one wishes to consider the effects of mental activity upon other parts of the body, or the effects of certain bodily functions upon mental activity, he may do so as legitimately as he may study the effect of exercise upon the heart rate or the effect of the heart rate upon exercise tolerance. True, his methods of study will vary according to his problem, but in each instance he is investigating the influence of one body function upon another. And if he is a well-trained investigator he will realize that other body functions which he is not measuring will, nevertheless, be influencing the two functions which he is studying.

The " mind-body " relationship is thus resolved to a " part body — part body " relationship, and the simultaneous interaction and mutual influence of different body parts and functions is taken into consideration.

Psychogenic Behavior. You have already learned that ordinarily we speak of two kinds of effector mechanisms — muscles and glands. Although these are the only two that are commonly considered, it is recognized by most writers that our thought processes and other mental activities themselves represent responses.

Although we do not know the essential characteristics of thought, we do know that it demonstrates two of the major characteristics of other responses. First, thoughts and other mental activities may be elicited by stimuli; and second, they may lead to other mental activities, to covert actions, or to overt actions. That is, like other responses, they may serve as stimuli for new responses. Mental activities, then, whatever their nature, may be regarded as responses which set up new stimuli.

These stimuli, arising in the cortex and possibly other parts of the cerebrum, might well be termed " cerebral." If then, we wish to describe behavior in terms of the source of the stimuli which initiate it, we might call much behavior " cerebro-genic." Such a term would be very broad, for it would cover all integrated or partial activities stemming from perception, memory, learning, ideation, and thought. Some such acts would be involuntary and largely unconscious, such as the effect of worry upon our facial expressions, our posture, and our viscera. Some would be voluntary and fully conscious, such as our keeping of a planned appointment, or making a telephone call. Other acts would border between voluntary and involuntary, and between conscious and unconscious, such as the tying of a shoe as we dress after an early rising, or the semiconscious flicking of an ant from the arm as we concentrate on some outdoor work. All, however, could be described as " cerebro-genic " behavior in the sense that they had been initiated by impulses arising as a result of cerebral activities.

If you look in a dictionary, for the term " cerebro-genic " you will look in vain. You will find a similar term, however, **" psychogenic."** Apparently the word " psyche " will always be with us, so we might as well make the best of it. Let us remember, however, that it, or its translation, " mind," typically refers to processes which involve the cerebrum and which we call mental activity. Then let us remember also that such activity may be conscious or unconscious, voluntary or involuntary, and that the behavior it initiates may be perceived or unperceived. With these cautions we may substitute " psychogenic " for " cerebro-genic " and be in little danger of difficulty.

From a logical point of view, the two terms should be identical in meaning. Perhaps this may come to be true in the future, but today " psychogenic " has a somewhat restricted meaning. It usually refers to those forms of behavior and bodily effects which are not clearly perceived by the individual and which have occurred rather automatically, in response to " mental conflict," to perceived or imagined threat, to potential frustration, or to unpleasant emotional ideation. Because of this restriction in usage, you will more frequently encounter such a term as " psychogenic disorder " than you will the broader term " psychogenic behavior." It should be clear that psychogenic disorders constitute merely a particular subgroup among cerebrally initiated events. Without question, however, they are an important subgroup and merit our attention.

In the thinking of present-day psychiatry and psychology, psychogenic disorders include a wide variety of behavior. Among children they include some instances of thumb-sucking, nail-biting, enuresis, over-aggressiveness, over-submissiveness, and, in fact, almost every form of deviant behavior for which no organic bases can be found. Among adults the range is even greater, presumably because life places more responsibilities upon them than it does upon children and because they have had more years in which to develop ineffective habits. Here, examples include those forms of mental abnormality, personality disorder, and physical disorder which have no known organic basis — the neuroses, the functional pychoses, alcoholism, drug-addiction, sexual perversions, and psychosomatic illnesses, as well as any marked differences in personality.

Such a broad list, and the phrase, ". . which have no known organic basis . . ," should serve immediately to arouse your suspicions. Today the term " psychogenic " is used as a sort of waste-basket by psychiatry, general medicine, and psychology. If the doctor fails to find a physical cause for a given complaint, it is easy to tell the patient, " It's all in your mind," and then refer him to a psychiatrist or a clinical psychologist. Thus, the designation is overused through ignorance. Furthermore, even though one's mental attitude may influence the course of a disease or of a behavior disorder, it does not necessarily follow that the disease or disorder is psychogenic in origin. We shall see in Chapters 23 and 24 that many disorders in mental activity may be occasioned by physical factors within the body and, thus, may be thought of as "**somatogenic.**" But even with the additional insight which research is daily bringing, the importance of psychogenic behavior will not be greatly diminished. Psychiatry and clinical psychology will continue to develop and their common tool,

psychotherapy, will continue to be used in attempts to alter mental activity toward a reduction of psychogenic disorder.

Psychosomatics and Somatopsychics. A good dictionary will define " psychosomatic " as an adjective descriptive of interrelationships between mental activity and other bodily functions. Such a term describes the essence, of course, of the material covered in this text, whether it be called physiological psychology or psychophysiology. Unfortunately, however, the common usage of the term " psychosomatic " does not follow this strict definition. Most commonly it is employed to designate a more or less chronic illness or physical disorder which is regarded (1) as of psychogenic origin, or (2) as significantly influenced by mental activity.

In the last several decades it has come to be appreciated that most disorders of our bodies belong to one or both of these classes. Thus, psychosomatic medicine has come to assume tremendous importance. As a student of psychology you should know something about this field, and it will be treated briefly in following sections. You should remember two things, however: (1) all psychosomatic problems are not medical problems, and (2) psychology is concerned with organismic behavior which involves interrelationships and mutual influences — not only psychogenic effects.

Let us consider a specific example of a psychosomatic problem — the interrelationship of mental work and tension in skeletal muscles. First, we place you in a comfortable chair, ask you to relax, and play softly for you some mildly interesting music. Unless the music has meaning for you — unless you react to it as pleasant, unpleasant, good, or bad — there will be little change in your muscle tension or in other bodily functions. Then we ask you to work a simple arithmetical problem " in your head." Immediately we would find a slight increase in muscle tension (and if we were measuring your heart rate and the amount of sweating from the palms of your hands, we would find slight increases in both). Next, we give you a more difficult problem and allow you only 2 minutes to solve it. Especially if you are strongly motivated to succeed in this task, we should find a marked increase in muscle tension. Then we give you another difficult problem, with time limit, but this time we play for you some loud, cacaphonous music to serve as a distractor. If your motivation is strong and the loud music really distracts you, there will be additional muscle tension increases with, perhaps, overt restlessness and facial or verbal signs of frustration.

All of these bodily effects, of course, are psychogenic; and, also, they are psychosomatic. Obviously, they do not at the moment constitute a problem in psychosomatic medicine. Less obviously, they involve only one side of the interrelationship. They neglect the influence of muscle tension on mental work. To demonstrate this aspect of the interrelationship we shall shut off the music and ask you to perform mental arithmetic of varying difficulty as you press with varying degrees of muscle tension against a foot-pedal to which is attached varying weights. We shall find that, for simple problems, some increase in muscle tension will facilitate your solutions of them. As the problems become more difficult and your own general tension automatically increases, the additional imposed tension will more quickly reach a point where it begins to interfere with your problem-solving ability.

In this latter experiment we have an illustration of the influence of gross bodily activity upon mental activity. There are countless such examples, and many of them are important if one wishes to understand human behavior. Some of them will be discussed in the next two chapters. All could be called “psychosomatic” if the accurate definition of this term were followed. But since the common tendency when employing this adjective is to emphasize the psychogenic aspect of the interrelationship, we frequently employ another adjective to emphasize the somatogenic effect on mental activity — that is, the term **somatopsychic.** The term is just as inelegant as “psychosomatic,” but both seem to be necessary in our current thinking. Both may be used appropriately if it is remembered that each refers to the influence of one bodily function upon another.

Finally, it may be pointed out that each adjective requires a noun; and if we can employ a given noun which specifies its adjectival qualifier, we often facilitate both thought and communication. With this purpose, this text occasionally will employ two nouns which have been used elsewhere, but are not yet in common usage: (1) **psychosomatics,** which shall connote all psychogenic changes in bodily functions other than the direct influence of one mental activity on another mental activity; and (2) **somatopsychics,** which shall connote all somatogenic changes in mental activities. Throughout this text, of course, we have been concerned with both of these subjects. Together, they constitute the true subject matter of physiological psychology. In the remainder of this chapter and in those that follow, we shall hope only to emphasize their importance in any attempt to understand human behavior.

Psychosomatic Disorders. We already have considered many situations in which cerebral action (including mental activity) has an effect upon other organs of the body. Perhaps the most dramatic among such psychosomatic effects are those that have come to be known as psychosomatic disorders or psychosomatic diseases. We find that most of them are intimately associated with emotional behavior and some may even be referred to as emotion-caused. Let us see what disorders fall in this class.

Many of these psychosomatic disorders are well known. Most of you have acquaintances who at some time or another have suffered from psychogenic varieties of diarrhea, asthma, hives, constipation, hay fever, peptic ulcer, or high blood pressure. A number of related disorders which also may be psychogenic are less frequently observed. Among these are angio-neurotic edema, which involves swelling in various parts of the body; Reynaud's disease, in which the blood supply to the extremities is interfered with or almost completely interrupted by vasoconstriction of small arteries and capillaries; hyperhydrosis, in which there is exaggerated sweating from the palms of the hands, the fingers, and the bottoms of the feet; Hirschsprung's disease, which is a specific form of constipation with marked relaxation of the lower bowel; mucous colitis, in which there is hyperactive peristalsis of the intestines, and in which occasionally the innervation is so great that one finds tonic spasms with so-called spastic colitis; and many others which occur even less frequently. Psychosomatic medicine today is extending this list and probably there are other disorders which will be included, such as some forms of heart, kidney, or gall bladder disease, many skin disorders, and many disorders of sexual function.

Many illnesses have been recognized for some time as " nervous " or " functional " disorders (in other words, those for which no organic bases have been found) . Chronic fatigue is a classic example of these functional disorders; enuresis is commonly so regarded; " nervous indigestion " has been experienced by most of you; and the relationship of insomnia to worry is well known. Less generally appreciated are the psychogenic effects on vision and other senses, on glandular and other biochemical inbalances, on the common " cold," the manner in which mental activity can influence the course of an organic disease such as tuberculosis or cancer, and the psychogenic factor in at least some " accidents." Also, and even less well understood, there is a wide class of disorders known as **hysteria** in which a partial or total dysfunction (such as blindness in one eye, anesthesia in a hand, paralysis of a limb, or partial deafness) may appear quite suddenly and may disappear just as suddenly only to be followed by another hysterical symptom.

For most of these psychosomatic disorders there is no known organic basis other than tissue changes initiated by mental activities. For some, the tissue change facilitates the development of a disorder, such as a stomach ulcer. Apart, however, from the apparent absence of organic basis, there is another essential criterion for designation of a disorder as psychosomatic. It must be demonstrated that the disorder can be intensified or reduced through psychological intervention (e.g., by psychotherapy or hypnosis).

Theory in Psychosomatics. In the remainder of this chapter we shall attempt to show that psychosomatic disorders are related to emotional behavior, to muscular tension, and to learning. They occur differentially in different individuals who are faced with emotion-provoking situations; and this differential occurrence in individuals usually is attributed to certain organic deficiencies, which might be called predispositions or **diatheses.** Some organ diatheses probably are hereditary, but without question many have been acquired.

We find little convincing evidence to support the current view that psychosomatic disorders occur differentially in individuals with different personality patterns. The very conditions that underlie a given psychosomatic disorder may, upon occasion, effect certain differences in personal-social behavior of a sort that is called "personality." Furthermore, the actual presence of a psychosomatic illness may alter the personality. We would hold, then, that in so far as different personality patterns may be found to accompany different psychosomatic disorders, both usually are the sequels to underlying physiological patterns. This point of view does not deny, of course, that certain habits or certain factors in the external environment may alter both personality and psychosomatic functions. It merely questions current statements that there is an "ulcer-personality" which is essentially different from, say, an "asthma-personality"; and questions that personality is, itself, an important factor in "organ choice" — i.e., in determining the specific nature of the symptom. Rather, both organic reaction patterns and personality patterns are conceived of as developing out of a common background of hereditary and environmental influences.

The Role of Autonomic Functions. In order to understand the manner in which emotion may initiate and aggravate physical dysfunctions, we must give additional attention to the specific functions of the two branches of the autonomic nervous system and to the phenomenon of parasympathetic overcompensation. If we considered separately each of the psychosomatic disorders, we should find the ANS involved in each of them. For our present purposes it will be sufficient to consider a few examples. Let us select the following:

(1) activity of the sweat glands, which are innervated solely by the sympathetic branch; (2) the secretion of HCl in the stomach, which is innervated solely by the parasympathetic branch; and (3) the mucosa, or mucous membrane lining of the lungs which is innervated by both branches of the ANS.

You already know that sweat changes are very labile indicators of SNS activity and that they may be recorded electrically. Some individuals, because of heredity or past experience, have extremely reactive sweat glands. Even in a resting state the electrical resistance of their palms will be low and, with any emotional situation which further activates the SNS, it will drop and they will sweat more profusely from the so-called emotional sweating areas, i.e., from the palms of the hands, the soles of the feet, the "armpits" or axillae and, in some individuals, at the groin and in some facial areas. Sweating from the hands is, of course, particularly noticeable. If such an individual finds himself under the influence of extreme anxiety or almost any profound emotion, his hand sweating becomes to him almost intolerable. It presents itself almost immediately at the beginning of any emotional change which involves the SNS and it is maintained throughout its course. In fact, almost the only situation in which such an individual has really dry palms is during restful sleep.

This condition of **hyperhydrosis** is typical in some individuals who are hyperemotional. Of course, it is accompanied by many other ANS changes. It may be helped by drugs or by surgery — by cutting the nerve supply to the most reactive sweat glands — but this does not change the remainder of the SNS functions which also are often hyperreactive. Nor does it alter greatly the emotional reactivity of the individual. The therapy of choice in this situation is psychotherapy. Emotional reactivity must be decreased through relearning — through new orientation to life situations. Then, and only then, will total SNS activity be reduced. For some individuals, psychotherapy may not be sufficient. Organ diathesis is too strong. For them, surgery or continuing drug therapy is indicated.

The secretion of HCl is selected as a second example of ANS function in psychosomatic disorder only because it is one of the few bodily functions innervated solely by the PNS. Alone, it has little significance. Together with other PNS innervated functions, it has tremendous importance.

As we have seen in Chapter 21, in connection with certain emotional behavior there is excessive secretion of HCl. Of course, this excessive secretion rarely occurs alone. Typically it is accompanied by increase in other PNS functions, of which we should mention, in particular, increased stomach motility and increased blood supply to the viscera. There is another change which we have not yet discussed. In connection with these events there often is a decrease in the mucous covering of the lining of the stomach. Now let us combine all of these events and see what happens.

Typically in fear or anxiety there is an inhibition of HCl secretion, an inhibition of stomach motility, and a decreased blood supply to the stomach wall. Under some sorts of emotion, (e.g., chronic resentment) , or as an aftermath of strong emotion (i.e., by parasympathetic over-compensation) , the picture may be reversed. Then there is excessive HCl secretion along with increased motility and increased blood supply, even to the point that minute hemorrhages may occur in the stomach wall. If, at the same time, the mucous substance which ordinarily lines the stomach has been decreased, we have the kind of a situation which can produce a stomach ulcer. There is rich blood supply to overactivity stomach tissue which then, with or without minute hemorrhages, becomes more sensitive to the action of HCl. In short, HCl under such a condition begins to consume the lining of the stomach, and an ulcer is precipitated. As has been pointed out, such events appear to occur rather directly in some emotional situations, such as resentment. In the more profound emotions, they seem to occur after some period of time and lead to the supposition that initial SNS activity is sometimes followed by a period of increased PNS activity which may be regarded as overcompensatory in nature. Much more research is needed before the course of these events can be more accurately described. But it is certain that the events leading to the development of an ulcer in the stomach or intestine are determined by excessive PNS activity; directly, or as a secondary phenomenon. As was the case with hyperhydrosis, the symptom may be treated with drugs or surgery — in this case by vagotomy, an operation that severs the connections of the vagus nerves to the stomach and intestines. But psychotherapy again is the method of choice. If, by re-education, the ANS activity can be decreased, then not only will the ulcer-tendency be reduced, but also the tendency to develop other psychosomatic disorders. In some cases, drug therapy or surgery is necessary.

Finally, let us consider the asthmatic individual — he who tends to have difficulty in breathing at certain times. Sometimes he is described as allergic to certain pollens or other substances. Sometimes no evidence of allergy can be found. But always we find that PNS innervation to the lungs is excessive. The mucous cells are induced to secrete and there is marked vasodilatation of the blood vessels with swelling of tissues. In short, the airways become congested and breathing becomes difficult. The classic method of treatment in acute cases is a quick injection of adrenalin — a powerful SNS stimulant. The change is dramatic. The mucous secretion stops, the blood vessels and their surrounding tissues constrict, and the patient again can breathe. If you ever have whiffed a " benzedrine inhaler " you have experienced the same kind of events in the mucous membranes of your nostrils.

Again the most effective form of treatment often is psychotherapy. Eliminate pollen or other forms of allergic substances and the patient may be helped. Sometimes drugs may be used effectively, but in this case surgery is impracticable. The branches of the vagus nerves that innervate the lungs are easily confused with those that innervate the heart. The most effective therapy is to re-educate the patient — to alter his habitual reaction patterns in such a manner that his parasympathetic nervous system will no longer overact to life stresses primarily or secondarily, for lungs or other organs.

The Role of Muscular Tension. We already have had occasion to distinguish between phasic and tonic muscular contraction. Whenever tonic contraction occurs we speak of tension as existing, or sometimes it is referred to as muscle tone. We have seen that most of the smooth muscles of the body have the characteristic of slow and long contraction and may be said to exhibit more or less continuous tone. In so far as they exhibit phasic contraction at all, the phases are long. The general pattern is one of slow increase or decrease in tone, since most of these muscles or muscle groups are innervated by the autonomic nervous system and are in a state of more or less continuous activity.

It is also possible, however, for striated or skeletal muscles to exhibit varying maintained states of tension. This phenomenon is typical, of course, in those muscles which aid us in maintaining our posture. They are referred to as the antigravity muscles because they oppose the force of gravity whenever we are in an upright position of any sort. During most of our waking hours, especially when we are not reclining, some or all of these antigravity muscles are in a more or less constant state of tension. Even then, however, there are marked individual differences of several sorts.

We are all familiar with the individual who appears to be tense most of the time. His movements are apt to be jerky, his face expressive and sometimes contorted, and he rarely seems to relax and take things easy. Somewhat less frequently we see the opposite extreme. We see a person who is so relaxed he gives the impression of being almost asleep. His arms swing freely and loosely from the shoulders, his movements are apt to be rather lethargic, his face muscles are relaxed and give the appearance of sagging or hanging loosely, and he seldom goes into action quickly. Most of us lie somewhere between these two extremes and some of us are capable of optimum differential relaxation.

Differential relaxation is a condition which, as we shall see, is well worth considerable effort to learn. In some individuals who are extremely well coordinated muscularly, it would seem to occur rather naturally; that is, without any conscious effort or learning. In many others, it is something that apparently does not occur naturally and a large amount of practice is necessary in order to achieve it. That it can be achieved has been demonstrated by much experimental work and by the reports of the Yogis of India.

The lack of differential muscle reaction is well shown in the act of writing. Tension in some muscle groups is necessary, of course, for this act. We must exert enough tension to maintain the grasp upon the pencil or pen we are using. Moreover, tension is involved in upper arm muscles in order to move the hand which actually contains the writing instrument. And, of course, we have tension in the antigravity muscles which maintain us in an upright seated position.

Beyond this necessary minimum amount of muscle contraction, however, most of us go much further. It is only necessary that we maintain a tight grip upon the pencil while actually writing a word; but measurements have shown that the majority of us maintain a grip in between the writing of words, or while we are thinking of what we are to write next. Sometimes we start the grip before we actually begin to write; often we maintain it between words; and once we have completed a sentence we occasionally maintain our tight grip on the pencil for a long period of time.

This lack of differential relaxation is not confined, however, to the hand and arm muscles. It will be noticed frequently that the individual who is not relaxed in the hand and arm when not engaging in actual writing activity is usually not relaxed in other portions of his body. He is apt to be sitting on the edge of the chair; he is apt to be engaging in restless movements with the legs, the feet, the unoccupied hand, or the head and neck muscles. In short, he might be described as rather tense throughout his entire body. Usually such individuals also find it difficult to relax when they retire. They admit to trouble in falling asleep and complain of feelings of tenseness in various parts of their bodies. If we were to measure action potentials of various muscle groups of the body in this kind of a person, we would find more or less continuous bursts of activity, which would continue as long as the individual was awake.

Actually, the proper way to put this kind of a statement would be in the reverse. We should say that an individual's condition of wakefulness continues as long as his muscular bursts can be recorded. Rather general relaxation in the striated or skeletal muscles of the body usually is a prerequisite of the condition we refer to as sleep; and it has often been shown in the laboratory that among the last muscles to relax before sleep are those involving the head, the eyes, the jaws, and other portions of the speech musculature. In fact, as long as we are awake, alert, and thinking, there is some degree of muscular tension in portions of our striated muscles.

Can we infer then that when sleep is said to exist there is no tension in these striated muscles? That hardly seems possible in view of the findings of many investigators that we characteristically move considerably during sleep. We engage in all sorts of restless turnings-over, etc., all of which presume some tension in the skeletal muscles. There is a definite relationship between wakefulness, consciousness, and muscular tension, however. In general, it may be stated thus: the more muscular tension, the more wakefulness; the more muscular relaxation, the greater the depth of sleep. The next chapter will consider sleep and wakefulness in greater detail. Now, we turn our attention to the psychosomatic aspect of muscular tension, to psychogenic influences on our skeletal muscles.

It should be emphasized that there are no entirely satisfactory techniques for the measurement of characteristic level of tension in skeletal musculature. Although we look at individuals and describe them as tense or relaxed, and although sometimes we are fairly sure of our judgments, the actual measurement of whatever it is we are judging is an extremely difficult problem. One way to get at it is by the technique of the rating scale; and if the ratings are made by several individuals, each of whom knows the rated subject very well in a number of situations, this technique can be quite reliable and should furnish a validation measure against which other techniques can be tested.

The desire to find objective methods of measurement, which do not depend upon past knowledge of the subject, has led to many individual attempts. The method of choice probably is the recording of action potentials from muscles, but no single muscle group has yet been found which accurately reflects tension in other muscle groups. In other words, patterns of differential relaxation are so great, even in supposedly tense individuals, that we cannot base our judgments on any one group of muscles. A combined measure from a number of simultaneously recorded action potentials from different muscle groups might give an adequate over-all result, but such a technique is rather laborious and impractical. Although the electrical resistance of the skin is known to markedly increase as an individual relaxes, and may be used with considerable validity for measurements of different degrees of relaxation for a particular subject at a particular time, so many other factors enter into electrical skin resistance change that this technique leaves much to be desired. Since the hand, and the pattern of muscular tension within it, seems to reflect tension in other parts of the body fairly well, it has been used by several investigators. Different techniques have been employed, such as grip pressure or pressure of the writing instrument during the act of handwriting. These techniques show promise and recently devised apparatus for their measurement is now being employed. Since tension is such an important component in our daily motor activity, and since it is a factor which enters into a number of personality traits, the designer of an adequate technique for the accurate measurement of individual differences in muscular tension levels will make an outstanding contribution to psychology.

The most important fact which we wish to emphasize at the moment is that whenever we experience profound emotion, tension in skeletal muscles also increases. The reason for this increase is unknown. Striate muscles are not innervated directly by the ANS. They seem to be innervated indirectly during emotional behavior, but we do not know the actual mechanism. Suffice it to say that if you become frightened or angry, not only your autonomic nervous system will respond but also your skeletal musculature.

As you already know, increases in skeletal muscle tension will facilitate your mental activities to a certain extent, but beyond that they will interfere with your efficiency. But such tensions have another influence which you may not realize. They actually contribute to your emotions.

This facilitative function of muscular tension deserves to be emphasized. It not only contributes to your sensory acuity up to a certain point; it not only contributes to distraction beyond a certain point; it also contributes to level of emotional behavior. In general, with increased emotion we are more tense muscularly, but, also, with increased muscle tension we are more emotional. Thus, any factor which increases muscle tension will also increase emotional behavior and other psychosomatic functions. Is it any wonder that some psychotherapists (including the Yogis) concentrate upon techniques for inducing muscular relaxation? How often have you, yourself, told a friend, " Just relax — everything will be all right "?

Muscular tension and relaxation are important in human behavior. We have much to learn about their control, but we know this. Any factor that will exaggerate tension in skeletal muscles will exaggerate psychosomatic problems. Any factor that will decrease tension in skeletal muscles will alleviate psychosomatic problems. There exists here a fertile field for research. In the meantime, psychotherapy, chemotherapy, and surgery, will attempt cures. In the meantime, also, let us consider learning as a factor in psychosomatic disorders.

The Role of Learning. First, let us review some of the fundamental concepts of the conditioned response which is one form of learning, and then consider some learned organismic patterns of response which are of wide-spread importance in human behavior. After an introductory course in psychology, you all should be familiar with the basic work of Pavlov and of many investigators who have followed in his footsteps. The early work of Pavlov and his colleagues who experimented on conditioned salivary responses in dogs, and the work of other Russian investigators on conditioned skeletal movements in man, discovered and well corroborated the basic principles of conditioned responses. In this work, however, the concentration of attention was on a particular response which was regarded as being of reflexive nature, that is, the salivary reflex, or the reflexive withdrawal of the hand or foot from an electric shock. It was not until some time later that the realization dawned upon workers that more than a particular reflex was being conditioned. Actually, the learning was organismic; it involved not only the reflex being measured, but a total response pattern in the organism of which the reflex was only a part.

Individual differences in conditionability of response patterns was another matter largely neglected by the Russian physiologists responsible for the early work in this field. It is true that Pavlov did discuss problems of individual differences, and he reported that his dogs roughly fell into two classes. One he referred to as the excitable class, which included those who were easily conditioned; the second he referred to as the inhibitable class, or those in whom conditioned inhibition was easily established.

The symbols **CS** and **UCS** have become almost standard in psychological literature. Although the term, **conditioned stimulus** (CS), has been questioned (and it would seem more appropriate to refer to it sometimes as the to-be-conditioned stimulus), it has been used so extensively that it is probably with us to stay. This is not a serious semantic problem as long as we clearly realize that the term has two distinct uses. It refers to a given neutral stimulus before any actual given conditioning has occurred (i.e., the to-be-continued-stimulus). It also refers to a stimulus which comes to elicit a given conditioned response.

An **unconditioned stimulus** for a given experiment is merely an effective stimulus. It is one which is effective for eliciting a certain response pattern, of which the experimenter may be studying only a small portion.

After a sufficient number of paired stimulations have occurred in which the neutral (CS) stimulus and the effective (UCS) stimulus have been presented in close contiguity, we then find that the response elicited originally by the UCS only can now be produced by presentation of the CS alone. It must be emphasized here, however, that the **CR (conditioned response)** is rarely if ever identical with the **UCR (unconditioned response).** Perhaps no clearer example of this fact can be found than the first experiment on conditioned fear, an experiment with which most of you are already familiar. A white rat was used as the conditioned stimulus, to which the original reaction of the child was an approach reaction, sometimes even involving petting of the animal. This stimulus was coupled with a sudden loud noise which elicited a startle reaction, the unconditioned response. After several paired stimulations the pattern of response to the white rat began to change; but never did the typical startle pattern occur to the CS. Instead, we find reported a marked variability in the behavior of the child. Always the pattern involved aversion. Sometimes it included semistartle and whimpering.

It is apparent from this brief description that the conditioned pattern of aversion and the total response pattern in this child were characterized by variability and by difference from the unconditioned response upon which they must be said to be based. These characteristics are often neglected in treatments of conditioned response principles, and yet they would seem to be the rule rather than the exception whenever we are dealing with conditioned responses. Once the response has been conditioned it will not be invariable. In fact, seldom will it occur in exactly the same manner to two presentations of the CS; and the more extensive our measurements of total behavior pattern, the more vividly is this fact demonstrated. Furthermore, rarely if ever, will the CR ever be the exact duplicate of the unconditioned response. Therefore, it cannot be considered as an exact substitute.

In Chapter 21 the organismic pattern of fear was discussed in some detail. Had the investigator in the above cited experiment attempted to measure autonomically innervated responses in his child subject, he no doubt would have found the entire SNS pattern which we called fear. He also would have found, however, that these responses were variable. They are influenced and modified by all the factors which serve to control and alter behavior — by the condition of the organism, its state of health, processes which are going on within it at the moment, by the nature of external stimuli which are reacting upon the organism at the time, and by the extent of excitation within the nervous system itself as a result of past experiences with the conditioned stimulus pattern. One factor, however, stands out as being less effective in altering or modifying these internal or external responses. That is the factor that we sometimes call volition.

In our first discussion of the autonomic nervous system and, in fact, in the earlier discussion of smooth muscle tissue in Chapter 5, it was pointed out that smooth muscles and vegetative organs in general are usually thought of as representing involuntary or automatic responses. The distinction between voluntary and involuntary muscles was questioned and the validity of referring to the autonomic system as an entirely automatic system also was thrown in doubt. Let us consider further the bases for those questions.

There are perhaps no more dramatic examples of the extent to which man can learn to control his so-called involuntary processes than some of the practices of Yoga. The Yogis are a Hindu sect who, by virtue of long training and long study, gain control over many portions of the body which most of us are unable to control. Apparently they become able to control musculature of the gastrointestinal tract to the point where they may inhibit or induce peristalsis in either direction. Thus, they may induce defecation or regurgitation almost at will.

The fact that man can learn to control voluntarily such supposed automatic activities is ample testimony that their control can be learned unconsciously. In other words, through unconscious learning, our past experiences often have effects upon our viscera.

Although many of the Yogic practices have been carefully observed and accurately reported, the actual learning process has seldom been carried out under controlled laboratory conditions. Certain attempts have been made in the psychological laboratory to place smooth muscle responses under voluntary control, and other instances may be cited in which other autonomic nervous system responses have come under voluntary control. Experiments have demonstrated, for example, the learning of voluntary control for the pupils of the eye, for the blood vessels of the hand, for pilo-motor erection, and for sweating. In summary, it may be said that there is ample evidence for the statement that we can learn to control smooth muscles.

These findings have vast importance for the student of human behavior. We all engage in a great amount of this kind of conditioned smooth-muscle behavior. Sometimes we regard it as voluntary, sometimes as involuntary, but always it will have the characteristics of conditioned responses to words or to other symbols. When we think of a situation which has produced fear in us in the past, this thought, or sometimes the image which we recreate of the situation, serves as the stimulus to elicit a true pattern of fear in us. Most frequently it is mild but, nevertheless, it legitimately can be classed on the anxiety-fear axis. We think of other situations and become angry or happy or excited; and still others cause us to become sexually aroused. But since the words, thoughts, and images themselves are reponses to other stimuli, we may think of much of this behavior as higher-order conditioning; that is, response patterns which are being elicited by stimuli arising from other response patterns. This is the basis for much of our behavior that we refer to as adjustment or as maladjustment. All are psychosomatic in nature.

Conditioned Patterns of Tonic Contraction. You have already learned that practically any of the phasic movements of the body can be conditioned to practically any stimulus, given the proper conditions. Although few experiments have been conducted involving the conditioning of tonic contraction patterns, there is every reason to believe that this occurs very frequently in man and in lower animals. Many examples in your own behavior will convince you of this generalization and all such conditioned behavior may be described as psychosomatic.

We have mentioned in preceding pages the tendency of some individuals to grasp a pencil firmly before they even start to write. In short, they are " set " to begin to write. The term " set " is even more commonly used for the runner. For him the signals " on your mark " and " get set " have definite meaning. He has learned through practice the most efficient kinds of neuro-muscular patterns to be used for such stimuli. They represent conditioned tonic contraction patterns. The first are less intense, less extensive; the second are more intense and extensive.

The term, " get set," is used in all sorts of situations. We are set to listen; we are set to run; we are set to throw; or we are set to kick. You may think of yourself in each one of these situations and actually set yourself for each one of these reactions. You will realize that in each case there are changes occurring in your postural background — in the pattern of tonicity in your striated muscles. Each of these changes presumably represents a conditioned response to certain variable stimuli and each prepares you for a definite type of activity which you have learned. In fact, when we say we are set for a particular act, we usually imply that we have learned that act and that we have learned a preparatory muscular tension pattern which is effective in preparation for that act.*

Consider now the relation of background muscular tension to what we call attention. Although attention involves a complex pattern of behavior, undoubtedly one of its major bases is tonic muscular contraction. Let us refer to the runner again. He is on his mark, with a rather definite muscular set. You would all agree that he is attending to the starter. He is anticipating the next signal of "get set." Without a period of previous learning he could not know, of course, that this would be the next signal and this is where the matter of verbal learning enters into the picture. But part of this pattern of attention is the underlying neuro-muscular tension pattern, and the manner in which it differs from his set to respond in different circumstances. Perhaps it will not be difficult for you now to understand that, through conditioning (one form of learning) we may get "set" for many different types of reactions — for running, for walking, for listening, for sweating, for ulcer, or for asthma.

The Treatment of Psychosomatic Disorders. Anything that we have to say about the treatment of psychosomatic disorders is solely by way of emphasis. Many times in this chapter we have stated that a psychosomatic approach to behavior emphasizes the effect of mental activity upon other bodily function. We have stated that surgery or drugs occasionally may be valuable in the treatment of dysfunction; but we have contended that regardless of hereditary or early constitutional predispositional tendencies, treatment directed toward relearning — call it psychotherapy, or what you will — usually is the most effective means of counteracting psychosomatic disorders.

The Importance of an Organismic Approach. What we have to say here also is repetition. We hope we have impressed you with the following facts: (1) the term "psychosomatic" should refer to interrelationships between mental activities and other bodily processes; (2) "psychosomatics," however, typically refers to psychogenic effects upon other bodily processes; (3) since the foregoing is true, it has become necessary to use the term "somatopsychics" to cover the instances in which noncerebral bodily functions alter mental activity; and (4) one cannot hope to understand human behavior unless he considers both psychosomatics and somatopsychics. In other words, he cannot understand himself or his fellows unless he takes an organismic approach. He must be able to envisage at the same time psychosomatic and somatopsychic events. He must be able to see them occurring simultaneously and with mutual interaction in a given organism. This is the true organismic approach to the study of man.

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* It should be mentioned that some preparatory muscular sets may occur apparently without learning, especially in newborn human infants or in lower animals. Preparatory sets for feeding in the human infant or in birds would be familiar examples. They represent what may be called instinctive sets.

Suggested Readings

1. Alexander, Franz, *Psychosomatic Medicine*. New York: Norton, 1950.
2. Dunbar, Helen Flanders, *Mind and Body: Psychosomatic Medicine*, 2nd Ed. New York: Random House, 1955.
3. Grinker, Roy Richard, *Psychosomatic Research*. New York: Norton, 1950.

23 *Somatopsychological Problems*

This entire book is concerned with psychological problems in which the initiating cause is physiological. But for emphasis we want to consider specifically such topics as the effects of drugs and oxygen deprivation. In the next chapter this approach will be extended further into the field of abnormal psychology.

FATIGUE

Anyone knows how it feels to be tired, and anyone knows that as muscular effort continues, especially if it is heavy, the capacity to do work declines. Common-sense usage lumps these phenomena together under the heading of "fatigue." But this results in considerable trouble when one tries to find a consistent definition of the term. No doubt most, if not all, students get tired of studying. But is this the same thing as getting tired from a few fast sets of tennis? Why should the stenographer who is "exhausted" from a day at the typewriter find ample energy for an evening of dancing? Perhaps it will be well to look at the meaning of fatigue more closely.

Work Decrement. A common-sense definition of fatigue relates it to observed reduction in work output as work continues. Industrial "work curves" are obtained by plotting output or rate of work against time of day. Sometimes these show a reduction in output toward the end of the day and this is ordinarily ascribed to fatigue. Actually, this is an erroneous approach. A reduction in output might occur for numerous reasons and an absence of a drop in output does not indicate absence of fatigue. Output may be maintained through increased effort. For crude practical work, this approach may be useful, but for careful analysis, practical or otherwise, sharper definitions will be required.

Physiological Fatigue. Since fatigue is most often studied in physiology, we shall first consider the meaning of physiological fatigue. Here the definition of fatigue is a reduction in response as the result of activity. For example, suppose that a muscle is stimulated over and over again by means of an electric shock. After a while, contraction is found to be less vigorous and eventually to cease. We assume that waste products have accumulated in the muscle, although we are not certain of the exact physiological changes involved.

Fatigue is not confined to muscles, nor is the muscle the controlling factor in continued contraction in the normal organism. If a muscle is stimulated via its motor nerve, it will eventually cease contracting, although the nerve is still conducting impulses. If the muscle is then shocked directly, it will again contract, thus demonstrating that fatigue has occurred at the myoneural junction rather than in nerve or muscle.

Except in sports, or extreme conditions such as combat, true physiological fatigue is seldom encountered. The "tired business man," or his employees either, for that matter, ends the day with no loss in muscular strength, tired though he may feel. In fact, workers often leave the job stronger than when they started in the morning! And certainly studying is not fatiguing, in the physiological sense of the word. But the student feels tired just the same! We are forced to conclude, therefore, that subjective feelings of fatigue and physiological fatigue do not necessarily go hand in hand. Neither, indeed, do performance and feelings of fatigue. We must, therefore, consider the psychological aspects of fatigue in their own right.

Psychological Fatigue. Feelings of tiredness arise in many situations, both active and sedentary. Tiredness may result from heavy labor or from simply sitting still too long. Tiredness is sometimes, indeed often, unpleasant, but by no means necessarily so. The pleasant tiredness following a good hike or a game of tennis is very familiar. So we must deal with two aspects of psychological fatigue: (1) feelings of tiredness, and (2) feelings of pleasantness and unpleasantness.

Feelings of tiredness can arise from sensations in fatigued muscles. No doubt this is a complex of pressure and pain, sometimes compounded into what we call ache. This is straightforward enough. Unfortunately, such feelings seem also to arise without any particular muscular exercise. Partly this can be ascribed to ache resulting from maintaining a set position too long — as in typing or studying at a desk — but partly these feelings are difficult to explain. Sometimes such feelings are called "mental fatigue," with the implication that they are based on processes different from those involved in muscular fatigue. Other terms are boredom and satiation.

The idea that there is a distinction between mental and physical fatigue is common. To some extent this is not unreasonable, and a consideration of our discussion of fatigue up to this point will show that tiredness can arise from mental as well as physical activity. Accounting for mental fatigue is not easy. Nerves do not fatigue very readily and, in any event, quiet mental arithmetic increases the bodily consumption of oxygen by a negligible amount. (One investigator expressed his disdain for this increase by pointing out that the energy for an hour's mental arithmetic could be obtained from half a peanut!) It has been suggested, and it would appear reasonable, that the development of " mental fatigue " and most especially unpleasant feelings of fatigue are related to conflict situations.[1] Pleasant tiredness involves no conflict. One has been doing something enjoyable and has not been forced into doing something he would very much like to avoid. On the other hand, unpleasant feelings of tiredness are related to conflict situations. One would like to stop what he is doing, but the requirements of the degree or the job make stopping impossible. From this conflict arise feelings of tension and tiredness. Here lies the explanation of our stenographer's behavior and of most industrial fatigue.

Fatigue in General. Our common sense term has turned out not to be so simple. In fact, one risks serious misinterpretation if he insists on calling all the phenomena we have discussed in the paragraphs above by the single term " fatigue." It is better to be more specific about what one means. **Work decrement** may be used for the straightforward reduction in observed work output. This has some practical use, but does not indicate the basis of the reduction. Physiological fatigue refers to the actual loss of ability to respond as the result of activity and is sometimes referred to as **impairment. Psychological fatigue** refers to the feeling of tiredness, no matter what its source. **Boredom** and **satiation** are applied where psychological fatigue has arisen in the absence of significant muscular activity. We have no accepted terms to distinguish between pleasant and unpleasant feelings of tiredness. It is best, therefore, to add these terms as modifiers of psychological fatigue, awkward though this may be.

SLEEP AND WAKEFULNESS

Cycles of activity and excitability are commonly observed in the animal kingdom. Sometimes, as in certain marine organisms, excitability and spontaneous activity vary with the tide. Sometimes, as in birds and mammals, excitability varies with the time of day. The living organism does not always show the same responsiveness to stimulation, but is likely to display a rhythmical waxing and waning of responsiveness. Some aspects of these changes in excitability are subsumed under the term **sleep.** Others, ordinarily less extreme, are referred to as changes in vigilance. We shall first discuss sleep and then extend our discussion to vigilance, or alertness, and the problems associated with it.

The Phenomena of Sleep. If we are to discuss the physiological patterns involved in sleep, we must first decide upon a criterion of sleep. When is an organism asleep and when is it not? Surprisingly enough, the decision is not always simple. There are several criteria and these are considered below.

The most striking characteristic of the behavior of a sleeping person is his relative unresponsiveness to stimulation. Although it is true that individuals differ not only from one another but from time to time in depth of sleep, some depression in responsiveness is always demonstrable. Early studies of depth of sleep concerned themselves with testing the magnitude of an auditory stimulus necessary to arouse the sleeper. The semipopular statement that the first two hours of sleep are the most restful depends heavily on such evidence.

Mere lack of responsiveness is not enough to define sleep. In hypnosis the subject may be quite unresponsive to certain types of stimulation. His reflex responsiveness need not decline, however, in the absence of specific inhibitory instructions. On the other hand, a sleeping person is not completely immobile or oblivious to stimulation. Sleeping persons change position about 30 times during a night's sleep, no doubt in response to patterns of stimulation in the skin, joints, and viscera. Also, responsiveness to specific stimulation may be maintained — the sleeper may be set to awake at the cry of the baby in the next room or upon some particular signal. Complete unresponsiveness would be indicative of **coma,** not sleep.

Another characteristic of sleep is the relaxation apparent in the sleeper's striate muscles, perhaps most obviously illustrated by the closing of the eyes. Although this relaxation varies from person to person and from time to time, an observer would not be inclined to consider a person asleep who is lying unconscious with rigid muscles, but rather as being in some pathological condition. Associated with the usual relaxation there is a general internal pattern which is describable as parasympathetic dominance. The heart rate declines, breathing becomes slower and deeper, bodily temperature declines, the skin resistance rises, and peripheral blood volume increases. In general, the usual picture is one of relaxation and reduced metabolic rate, with restorative processes outstripping the destructive ones; and, typically, the majority of these changes have occurred just before sleep and they are maintained during sleep.

Most recently studied have been the changes in the EEG associated with sleep. It may be said that these changes are similar to those found when a person is placed under an anesthetic. Figure 23.1 illustrates the shift from alpha rhythms, through delta rhythms, to the random electrical activity and spindle bursts of the thoroughly asleep person. Upon awakening, the changes are reversed. When first studied, interest was directed toward discovering the changes in the EEG with sleep. Now, the situation is reversed. The EEG pattern is coming to be accepted as the criterion of sleep. No matter how relaxed a person may appear, no matter how deep his breathing and how slow his heart rate, if his EEG displays alpha rhythms he is awake. As we shall see in a few paragraphs, this EEG definition of sleep and wakefulness is used in the study of brain activity under circumstances which do not permit the application of any other criterion of alertness.

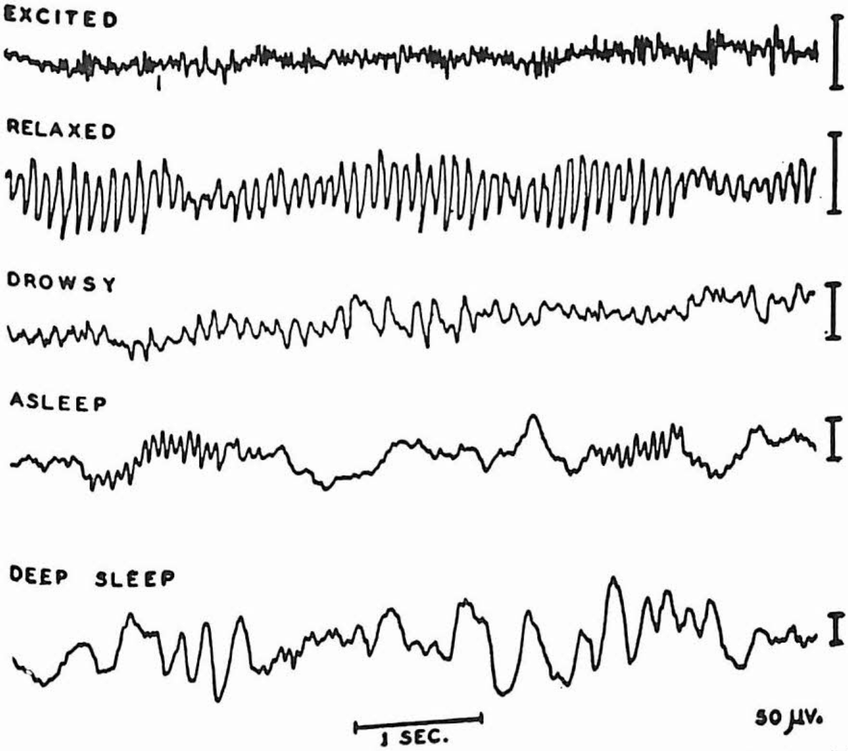

FIG. 23.1. Changes in the electroencephalogram (EEG) associated with sleep. (From Jasper.[2])

Sleep Rhythms. A characteristic of sleep is its periodicity. If we confine our attention to man, we find that this periodicity changes during the life of the individual. The newborn infant is said to be polyphasic in sleep, although it might be better to say that he is generally asleep but awake periodically! A total of about 2 hours out of the 24 is spent in wakefulness and these are spent largely in eating. We shall have occasion to note further evidence in later sections that sleep is the basic condition and that wakefulness requires arousal of some sort of alertness mechanism. In the case of the infant, hunger, a sharp pin, or other source of stimulation is necessary for wakefulness.

Most human organisms gradually develop a monophasic sleep cycle. No doubt this is to a considerable extent learned, and persons maturing in a society which practices the siesta will develop a biphasic pattern. Even in our own society, with its prevailing monophasic pattern, considerable divergence is found. Not only do many adults nap, but also the requirements of shift on which one works, or the late hour requirements of an enthusiastic night club life may lead to quite different timing of sleep and wakefulness patterns in different persons. Although discussion of the problem is beyond the scope of this book, it may be noted that the establishment and change of such cycles of wakefulness are of importance in planning shifts in industry or watches at sea.

Effects of Prolonged Wakefulness. There is no doubt that the more complex organisms at least require a certain minimum of sleep. If experimental animals are kept awake for long periods of time, they will eventually collapse and die. Under most circumstances, of course, an organism will fall asleep of necessity before such a fatal termination. In man, a good deal of the sleepiness which he experiences is probably the result of learned rhythms rather than sheer necessity. However, prolonged wakefulness very definitely leads to feelings of tiredness, dry and heavy eyelids, and so on, but it is difficult to demonstrate objectively any reduction in ability. Even when extremely tired, the organism can arouse itself to meet a particular challenge, and short objective tests of tapping, visual acuity, and the like are simply not useful indicators of impairment. There is evidence that more complex activity is altered.[2] After 72 hours of wakefulness subjects may see double, forget events which have just taken place, or hold books upside down in an attempt to read them.

Despite the deleterious effects of prolonged wakefulness, it is difficult to decide just what amount of sleep is really necessary for maximum efficiency and well-being. No doubt there are considerable individual differences and the conventional 8-hour recommendation is at best an average. Some persons require more — perhaps a good deal more — while others are content with less. This is partly a matter of how much discomfort one is willing to endure. College students are less upset, apparently, at feeling sleepy than they will be after graduation. At a time in life when it is likely that they require more than they will 10 years later, they characteristically sleep fewer hours. In any event, a normal person must suit his sleeping time to his individual requirements.

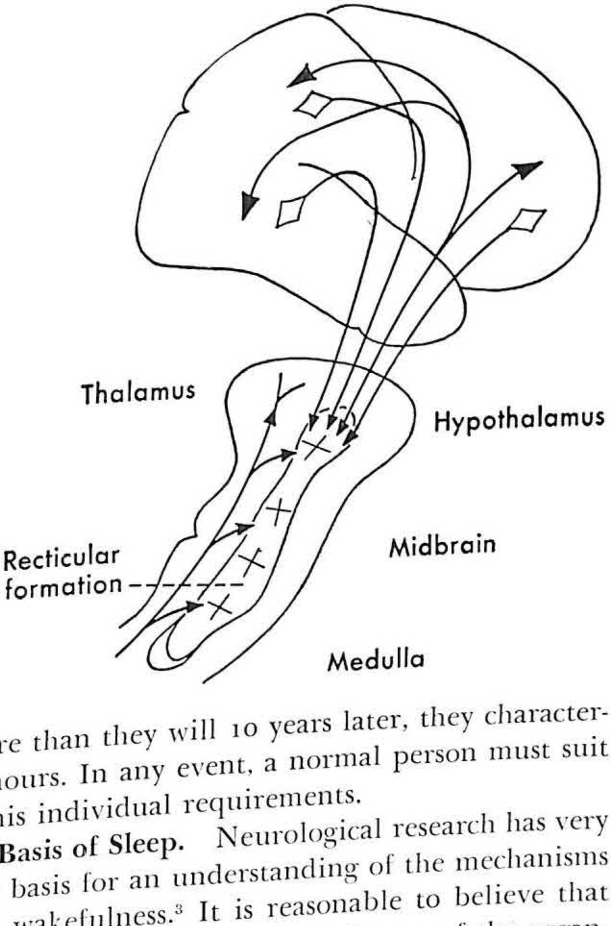

FIG. 23.2. Excitatory recircuits between the reticular formation and the cerebral cortex. (After Magoun.[4])

The Physiological Basis of Sleep. Neurological research has very recently provided the basis for an understanding of the mechanisms underlying sleep and wakefulness.[3] It is reasonable to believe that sleep is the basic state, wakefulness the activated state of the organism. In brief, if the wakefulness or arousal mechanism is destroyed or inhibited, the organism remains in a condition of sleep. Let us turn to the details of this mechanism.

Figure 23.2 shows the important structures of the arousal mechanism. The motor functions of the reticular formation were previously discussed in Chapter 18. In Figure 18.4 the upper portion was labelled with a plus sign and is excitatory. The caudal portion was marked with a minus sign because it has inhibitory or suppressor functions. With these latter we shall not at the moment be concerned. The reticular formation sends fibers broadcast to the cortex by way of the thalamus. It receives fibers from the various afferent systems and from the cerebral cortex. It, thus, has a feedback relationship with the cortex. Also involved in the arousal mechanism are the hippocampus and interconnections between the hypothalamus and the cortex (by way of the thalamus).

If the arousal mechanism is activated, the EEG shows the characteristic waking pattern. If this mechanism is destroyed, the EEG shows the pattern found in sleep and attempts to arouse the organism by intense stimulation produce effects lasting for only a fraction of a minute. It is interesting to note that these results of animal experimentation are in agreement with clinical evidence obtained by the study of patients in whom various parts of the arousal mechanism are disordered.

If sleep is, in effect, an absence of wakefulness, the problem remains of accounting for the termination of wakefulness in the normal animal. The answer is not completely available. There are several factors which must be considered. First of all, it has been suggested that the waking organism gradually builds up fatigue products which, circulating in the blood, in effect anesthetize the CNS. Despite the existence of a name, " hypnotoxin," there is no good evidence that such a specific product exists, although the constituents of the blood may play some role. Another factor is habit. Here we are on more solid ground. Sleep habits are very important in determining the onset of sleepiness. Not only is the time of sleepiness subject to habituation, but also the circumstances — the city dweller who cannot sleep in the comparative quiet of the country is by no means confined to fictitious anecdotes. Such factors as the ritual — tooth brushing, bathing, and even refrigerator raiding — which may ordinarily precede sleep also must be included here.

A third factor is afferent neural activity. It has been suggested that the feedback from one's muscles, for example, is of importance in maintaining wakefulness. No doubt this is true, but proprioception is not unique. Reduction of stimulation results in a reduction of afferent impulses to the reticular formation. Shutting the eyes, finding a quiet environment, relaxing the muscles, these all contribute to sleep. Their effect, however, is best understood as being upon the arousal mechanism, for, as pointed out before, afferent stimulation will not lead to continued wakefulness unless this mechanism is intact.

A last factor needs consideration. From time to time it has been suggested that, in addition to (in earlier work, in place of) the arousal mechanism there is a "sleep center." Stimulation of the anterior hypothalamus can lead to inhibition of the arousal mechanism and consequently to sleep. Perhaps there is a reciprocal relation, such that sleep or wakefulness depends upon a balance between the arousal mechanism and an inhibitory center.[4] More evidence is needed here.

Alertness. We have been discussing sleep and wakefulness almost as though they were mutually exclusive states, with no gradation of either. This is obviously untrue. Not only does depth of sleep vary, so that the sleeper can be aroused more readily at some times than at others, but also dreaming and bodily activity during sleep indicate that considerable neural activity can go on. On the other hand, there are equally apparent degrees of wakefulness. At times the organism seems to be highly responsive, alert to the world about it. At other times, responsiveness is lower, even though one would not consider the organism to be asleep. These apparent differences in responsiveness arise from two somewhat different sources. First of all, there is a general level of alertness — of vigilence, as it is sometimes called. Second, there is more or less selectivity in what is responded to, even given a certain level of alertness. Thus, someone engrossed in reading a fascinating book may not hear the doorbell. He is awake, perhaps very alert, but we say that he is paying attention to the book. This selective aspect of alertness is extremely important in behavior and depends upon the neural patterns of the moment as determined by motives and past experience. Our concern in this chapter is with general alertness and we shall not pursue attention further.

Since there are degrees of alertness, it follows that a reasonable control of general level can be accounted for as a balance between two mechanisms, one an arousal system, the other an inhibitory system. When we discussed the sources of sleep, it was suggested that this dual control is a possibility. The arousal or alertness mechanism is much the better understood, as we have already indicated in the discussion of sleep.

Another aspect of this mechanism, only briefly noted previously, needs emphasis here — the diffuse nature of the cortical effects of arousal activity. The great afferent systems, particularly those of somesthesis, have a dual central effect, possibly based upon separate fiber systems, possibly upon collaterals from a single system, such that activation of the arousal mechanism and specific patterned activation of the sensory areas of the cortex are to some extent separate functions. The sensory areas may influence the arousal mechanism only indirectly by way of the sensory "association" areas. These association areas send fibers to the arousal mechanism. In the absence of the arousal mechanism, specific patterned activity occurs in the cerebral cortex following sensory stimulation, but the organism is not normally aroused.

In addition to the evidence of alertness obtainable from the EEG and from responsiveness to stimulation, human subjects can report upon awareness. Admittedly, research upon consciousness has serious methodological difficulties. However, neurophysiologists have been concerned with this problem and the working out of the arousal mechanism has brought it to the fore. It may be said that sustained awareness, or consciousness, depends upon the arousal mechanism. We may tentatively conclude that awareness of specific sensory or perceptual patterns depends upon a background of general activity which is in turn dependent upon the activity of the arousal mechanism. This is speculative, but is not incompatible with the evidence available.

DIET

It is often heard that feeding a dog " red meat " will make him vicious. And primitive men felt that consuming different kinds of flesh would endow the eater with the qualities of the animal from which the flesh had come. It is doubtful that you believe these things, but perhap you feel that fish is " brain food," or even that eating too much meat may make you irritable. Perhaps such ideas seem rather ridiculous. But, after all, the body is composed of materials which must have been obtained by eating and, though the organism can alter what it eats and can even synthesize new molecules, there are certain amino acids, vitamins, minerals, and so on which must be in the diet for survival. It is not unreasonable to ask whether or not absence or excess of one or another of these materials might not influence behavior in specific ways or whether the total energy available from ingested foods might not be important.

Caloric Intake. The energy contained in food is measured in large calories, each **calorie** being the heat necessary to raise a kilogram of water from 0 to 1° C. In the body, the energy is released by oxidation. For survival, an organism must take in enough calories to make up for what is lost in maintaining bodily temperature, doing work, and carrying out its other metabolic processes. In some animals, metabolism is so rapid that starvation is very rapid. The hummingbird for example, must actually hibernate each night since otherwise it would starve in a very few hours. In man, death from starvation may require 30 or more days and existence in a semistarved condition may be almost indefinite. The loss of weight during starvation is not evenly divided among the different organs. An animal which has lost virtually all its fat and perhaps 60 percent of its liver weight, will have lost perhaps only 2 percent of its heart weight, and 3 or 4 percent of its nervous tissue weight. Brain function as measured by tests of ability seems to show but little impairment until starvation is far advanced.

Of considerable social importance is the effect of prolonged restricted caloric intake. Although casual observation indicates that apathy, lack of motivation, and preoccupation with the essentials of existence are the usual results of prolonged semistarvation, the clearest results arise from work on conscientious objectors during World War II.[5] The experimenters were able not only to control the diet and activity of their subjects, but also to keep them under constant test and observation. The most important results for our purposes were the developing lassitude coupled with irritability, the maintenance of capacity but not the inclination for mental tasks, and the intense preoccupation with food.

Specific Dietary Factors. We have already had occasion to mention the deleterious effect of Vitamin B_1 deficiency on cerebral function. In severe cases, deficiencies may result in actual lesions in the CNS. The other vitamins of the B complex are also necessary for proper CNS metabolism. Considerable attention has been paid to the study of vitamin deficiencies in World War II prisoners of war camps, especially those in Siam where 80 percent of the British prisoners succumbed.[6] The diet was very deficient in the B vitamins and a wide range of physiological and psychological symptoms was noted. It is difficult, however, to separate the effects of vitamin deficiency from those of the low protein diet and of chronic dysentery. Add to these the misery of prison camp existence and forced hard labor and the specific effects of vitamin deficiency are very hard to evaluate. Some experimental evidence is available which indicates increased psychoneurotic symptoms in Vitamin B_1 deficient young men and mental hospital patients. There is little reliable evidence, however, that increasing the vitamin B intake above a normal level has any psychological effect, either on personality or intelligence, although the optimum intake differs from person to person. The involvement of vitamins other than the B complex in determining personality is even less direct. It would appear reasonable to conclude that severe vitamin deficiency will affect behavior, not by some specific route, but because the organism is very sick. When the deficiency is for some factor especially involved in neural health, such as the B vitamins, CNS symptoms may appear rather prominently.

Recently there has been considerable interest in glutamic acid which is one of the **amino-acids.** The amino-acids are the building blocks which combine to form the proteins and several of them are indispensable in the diet. Since glutamic acid is important for cerebral metabolism, it has been suggested that increased glutamic acid intake will improve cerebral function. A large number of studies have been reported in the literature which purport to test this hypothesis, both with animals, rats in particular, and with humans. Where the controls have been adequate, the results have been almost universally negative. Nor has glutamic acid been unequivocally shown to have any effect upon epilepsy. These negative results are not surprising, since glutamic acid can be synthesized by the body and need not be in the diet for normal growth.

Various minerals are required in the diet, but the effect on behavior of deficiencies and excesses of these is relatively unknown. It has been suggested that a lack of calcium will lead to irritability, a result which would be predicted from the threshold-raising effect of calcium on neurons. In any event, it is the very rare American diet which lacks a minimum quantity of minerals, with the possible exception of iodine. As a result, even though it is possible to demonstrate changes in activity level in laboratory rats by manipulation of the mineral intake, genuine mineral deficiencies are infrequently encountered among the general population. The effect of severe change in the salt balance needs mentioning, however, since salt lack is a likely result of exercise in hot climates. The term "tropical neurasthenia" has been coined to denote the **syndrome** of apathy, weakness, cramps, headaches, and gastrointestinal upset which result from this condition.

Whether the fat, carbohydrate, and protein balance is important in determining personality and efficiency of behavior is a moot point, no matter how desirable a high-protein diet may be for physical well-being. Since blood sugar level is maintained at a high level for a longer time after a protein meal than after a carbohydrate one it is possible that a loss in energy at mid-morning and mid-afternoon could be prevented by a high-protein breakfast and lunch. This could scarcely be termed a direct dietary effect, however. In the same moot category is the question of animal versus vegetable foods. It may be doubted most seriously that any personality differences which might occur between high-vegetable and high-meat eating groups can be ascribed to the diets. The question of the nutritional value of one or another diet is best left to the physiologist.

One further suggestion merits consideration. It is possible that there are fairly large individual differences in dietary requirement. If this is so, then unsuspected deficiencies might exist in many persons and contribute to their modes of behavior. Future research may serve to clarify this issue.

COMMON DRUGS

There are many substances which can be classified as drugs. Any extensive consideration of drugs is out of the question in a text on physiological psychology. There are a few substances, however, which are in such universal use that they are of considerable interest in daily life. This is particularly true of those drugs which are constituents of common beverages, for example ethyl alcohol and caffeine, or which are in general use without the benefit of medical advice. Among these latter substances are aspirin and, less frequently but more importantly, the various derivatives of morphine. All furnish excellent examples of somatopsychological effects.

Ethyl Alcohol. Among the alcohols, we shall confine our attention to ethyl alcohol, the one found in so-called alcoholic beverages. Other common alcohols, such as methyl alcohol (methanol), which is in widespread use in antifreeze compounds, and the higher alcohols are so toxic as to be considered out-and-out poisons. Ethyl alcohol is obtained from the fermentation of starches and sugars, such as corn or molasses. The source has no bearing whatever on the chemical characteristics of the resulting alcohol. Pure ethyl alcohol has virtually no odor or taste so that any differences in flavor among alcoholic beverages result from the various other compounds which may be present. The amount of alcohol in a given beverage is ordinarily designated by "proof," which in American usage can simply be divided by 2 to give percent by volume. Thus, 100 proof is 50 percent alcohol by volume, and a wine with 20 percent alcohol is 40 proof.

Since the ultimate effect of drinking an alcoholic beverage depends upon the concentration of alcohol in the tissues, and since this is determined most importantly by the amount ingested, the amounts of alcohol in some common beverages may be of interest. Plain whiskey contains 43 to 50 percent alcohol. A highball containing 2 ounces of 100 proof whiskey contributes an ounce of alcohol to the drinker. Fortified wines contain approximately 20 percent alcohol, so a 4-ounce glass contributes almost as much alcohol as the highball. Beer varies in alcoholic content from state to state, but if we take 4 percent as a reasonable figure, a 12-ounce glass of beer contains a ½-ounce of ethyl alcohol. Cocktails may be either weak or strong depending on proportions. If we consider 50 proof as typical, a 3-ounce cocktail contributes a ¾ ounce of alcohol.

Before ingested alcohol can reach the tissues, it must be absorbed. Absorption rate varies as follows: (1) with concentration, the stronger the drink the faster the absorption; (2) with content of the stomach, absorption being the most rapid from an empty stomach, slowest in the presence of butter fat; and (3) from person to person. Beyond this, there will be considerable individual variation in effect, partly because of body weight, since the larger the person the more alcohol it takes to reach a given level of concentration in the tissues, and partly because of biochemical differences. Figure 23.3 is a semi-popular representation of the over-all effects of various doses of alcohol, given in terms of blood alcohol. If anything, these figures, which are not grounded in experiment, are too high.

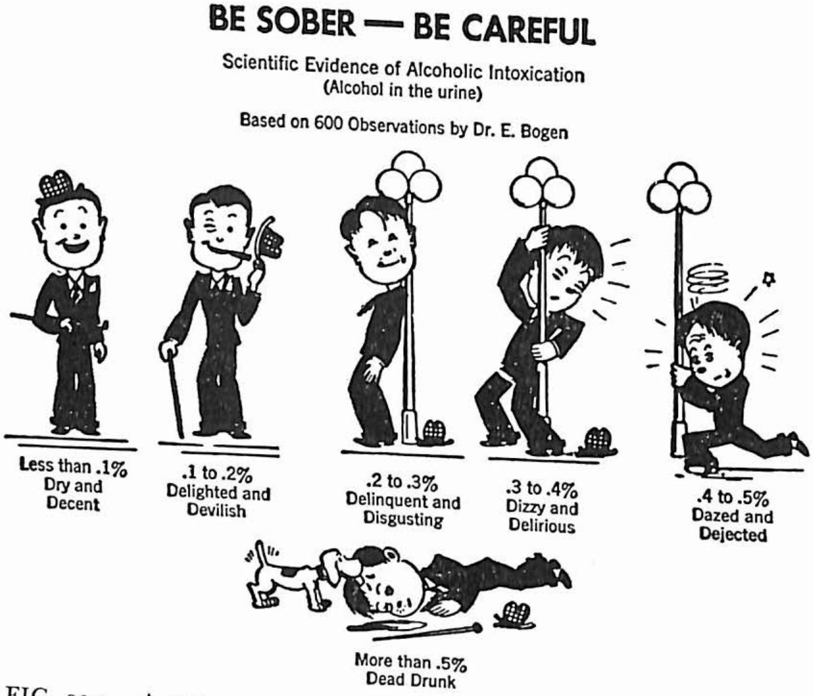
BE SOBER — BE CAREFUL
Scientific Evidence of Alcoholic Intoxication
(Alcohol in the urine)
Based on 600 Observations by Dr. E. Bogen
Less than .1% Dry and Decent
.1 to .2% Delighted and Devilish
.2 to .3% Delinquent and Disgusting
.3 to .4% Dizzy and Delirious
.4 to .5% Dazed and Dejected
More than .5% Dead Drunk

FIG. 23.3. A cartoon representation of the effects of blood alcohol on the behavior of the average person. (From Husband.[8])

Laboratory investigation of the behavioral effects of alcohol is quite clear in pointing to the conclusion that alcohol is a **narcotic** and reduces the efficiency of virtually every psychological function studied. This depressing effect is noted even for small doses and even though great care is used to mask the flavor of the alcohol. This narcotic effect, in a real life situation, may be obscured by the greater feeling of competence which may result from partial narcotization. Thus, an extremely tense person may sing better or make a better speech when slightly intoxicated than when sober, but still he is performing below his ultimate capabilities. If he could perform sober, he should be able to do even better. Unfortunately, as one becomes intoxicated his opinion of his condition does not keep pace with his actual decline in efficiency. Confidence may, and usually does, actually rise while competence is falling.

Longer-term effects may have important sociological consequences and a great deal of material, not all scientific by any means, has been published on alcoholism. In a text on physiological psychology, we can only indicate a few important points. In the first place, there is no doubt that habitual overindulgence in alcohol may have serious consequences for the addict, both in terms of his adjustment to life and in terms of his physiological well-being. Actual neural lesions can be observed in really bad cases and temporary psychosis is a common result. Recently, there has been some indication that much of the damage attributed to alcohol is indirectly caused by the displacement of necessary foodstuffs from the diet. Alcohol has a fairly high caloric value and the alcoholic does not eat enough other food to give him the necessary vitamins and other essentials. Vitamin B_1 deficiency seems to be the most important consequence. On the other hand, there is evidence that the craving for alcohol is related to vitamin deficiency. Bolstering the diets of alcoholics with the necesary vitamins may actually reduce their desire for alcohol. It is also possible that an excessive requirement for vitamins, as the result of individual biochemical differences, may lead to alcoholism. On the other hand, it seems very likely that much alcoholism is learned, not based on genetic differences. After all, a narcotic such as alcohol does reduce tension and anxiety and acts which result in such reduction are apt to be repeated. Especially if the problems of life are difficult to face, the blunting effect of alcohol may make them seem less important.

A final word should be said about the action of alcohol on the CNS. In keeping with other general anesthetics, alcohol tends to affect the more rapidly oxidizing portions of the CNS first. Thus, the cerebral cortex is pretty well narcotized while the medulla is still able to sustain the vital reflexes.

Caffeine. Caffeine is present in a number of popular beverages — tea, coffee, cocoa, and certain soft drinks. Table 23-1 shows the average caffeine content in grains of tea and coffee. Especially now that the " coffee break " has become a part of American culture, it is in-

Table 23-1. Caffeine Content of Coffee and Tea

	Grains of Caffeine
Average cup of hot black tea	1.5
Average after-dinner cup of black coffee	1.5
Average glass of green iced tea	2.0
Average cup of coffee with cream	2.5

teresting to ask whether or not drinking a beverage containing caffeine has any effects over and above those provided by the flavor.

In assaying the results of research on the psychological effects of caffeine, it is well to remember that there is a considerable tolerance factor. Not only do individuals differ in reactivity, but also repeated daily doses of coffee lead to the development of a tolerance so that the same dose produces fewer and fewer effects. Caffeine can be administered in tablet form, or in a more "normal" way in coffee or tea. No doubt results based on doses of tea or coffee are confounded by the effects of flavor, a warm drink in the stomach, and the associations, pleasant and unpleasant, which the subject may have between these beverages and past experience.

Careful experimentation has yielded fairly consistent results. Activity is ordinarily speeded up, at least with a moderate dosage of about 4 grains. Among the functions improved are reaction time, speed of tapping, typewriting, and color naming. Steadiness is, however, impaired. Strangely enough, small, 2-grain doses may retard the above functions instead of speeding them up. Large doses, especially continued over a period of time, will interfere with sleep, produce nervousness, and may lead to indigestion. The exact dosage which will lead to these effects depends upon the tolerance of the individual subject. Whether or not caffeine improves reasoning ability as well as the simple activities is not known.

Tobacco Smoking. The heading of this section, tobacco smoking, is used in place of " nicotine " because the effects of smoking are not solely the result of nicotine intake. Indeed, although an infusion of tobacco will contain nicotine in an amount depending upon the source of the tobacco, tobacco smoke probably contains little nicotine. Nicotine is altered through combustion largely to pyridine, which is not as toxic as nicotine. We shall confine our attention to the effects of smoking, leaving aside the question of the exact combustion products involved.

There is really not much information of a controlled sort concerning the psychological effects of smoking. Physiologically it is known that smoking in those habituated to it serves as a temporary stimulus for increased SNS activity. Vasoconstriction occurs in peripheral circulation, the heart rate is increased, and blood sugar is released. Such changes probably explain, in part, the "lift" that chronic smokers, and cigarette advertisers, claim. For nonsmokers, however, the autonomic effect is even more extensive and may include marked parasympathetic responses such as excessive salivation and nausea.

Evidence is lacking concerning some arguments for or against smoking. For example, in spite of the common practice among coaches of athletic teams to prohibit smoking among their players, there is no good evidence that adjustment to exercise is affected by smoking. The recent contention that smoking is important in the causation of lung cancer and heart trouble cannot yet be evaluated. Research here sems to suffer from the same difficulty which has beset some psychological research; namely, that if one compares persons who smoke with those who do not, he introduces factors which are perhaps correlated with the taking up of the smoking habit, but which are largely unknown. It is possible to show, for example, that smokers do not make as good college grades as nonsmokers. That this result is due to the smoking is much less likely than that smokers tend to be persons of greater social activity, hence less oriented toward studying.

There is one study on the psychological effects of smoking which was able to separate the effects of smoking from the effects of the smoke, and which eliminated the uncontrolled differences between smokers and nonsmokers. This study used blindfolded subjects, both regular smokers and nonsmokers, and introduced the ingenious control of comparing smoking with nonsmoking sessions by having the subjects smoke air heated by a heating coil during the nonsmoking sessions. If a little tobacco smoke was blown about the room, the subjects could not tell whether they were smoking tobacco or hot air, and so the factor of suggestion was ruled out. The results are summarized in Table 23-2. It will be seen that there are some differences between the smokers and nonsmokers. In general, however, the effects are not very large. These results need further investigation of an equally well-controlled sort. In any event, neither outstanding benefit nor impairment is indicated. Quite untouched by this type of study, of course, is the effect on the smoker of going through the ritual of smoking. It is possible that some of the relaxing and steadying effect many smokers notice arises from the ritual rather than from the contents of the smoke, or perhaps to some extent from the rhythmical movements of smoking. Gum chewing or rhythmic foot tapping can be shown to have such a steadying effect also.

Table 23-2. Effect of Smoking on Efficiency

[From Hull]

		Nonsmokers Test Number			Habitual Smokers Test Number		
		1	2	3	1	2	3
Neuromuscular Processes	Pulse Rate	+ 12.63	+ 3.07	+ 2.74	+ 8.09	+ 7.25	+ 4.52
	Hand Tremor	− 29.80	− 31.20	− 23.20	− 38.30	− 22.30	− 4.80
	Tapping	+ 1.35	− 1.14	− 0.39	− 1.40	− 0.90	+ 0.50
	Muscular Fatigue	+ 32.20	+ 10.10	+ 6.20	+ 9.60	+ 3.70	− 12.50
Sensory-Motor Processes	A-test, Speed	—?	—?	—?	+ 0.10	− 1.20	− 1.40
	A-test, Accuracy	—?	—?	—?	+ 22.20	+ 32.10	+ 38.80
	Reading, reaction time	+ 1.08	+ 0.05	+ 0.54	+ 1.20	+ 0.05	+ 0.85
	Learning, reaction time	+ 4.41	+ 0.44	+ 3.65	+ 0.70	+ 0.35	+ 5.55
Higher Mental Processes	Adding, Speed	− 2.94	− 3.43	− 2.96	+ 3.00	+ 4.58	+ 6.55
	Adding, Accuracy	− 26.50	− 36.00	− 17.50	− 2.40	− 3.40	+ 1.20
	Memory Span	− 6.38	− 2.53	− 6.46	− 5.22	− 1.83	− 3.95
	Rote Learning	+ 8.02	+ 4.33	+ 3.61	− 10.10	+ 0.20	− 6.20

Such explanations would seem more probable for the casual smoker who does not inhale. However, reports from heavy smokers who habitually inhale suggest widespread psychological effects which become most apparent when smoking is voluntarily or involuntarily prevented. In either case, general irritability and irascibility seem to be increased and the craving to smoke may lead to atypical behavior. Although the symptoms are not as extreme as those following withdrawal of drugs from a drug addict, they leave little doubt that for the heavy smoker who inhales, the physiological equilibrium has been so altered by smoking that it is temporarily abnormal in its absence. Evidence is still lacking on the nature of these changes.

Aspirin. Aspirin is actually a trade name for acetylsalicylic acid. The pharmacologically active portion is the salicylic acid. Physiologically aspirin is essentially an **antipyretic** (i.e., it lowers fever) by action on the hypothalamus. It also reduces the pain of some headaches, perhaps by action on the cerebral blood vessels, and is important in the treatment of arthritis. Its effect on cutaneous pain is in doubt.[7] Individual differences in response to aspirin are considerable, and doses above 10 grains or so involve some risk. With these physiological effects, at least one of them being directly upon a brain structure, it seems likely that there should also be psychological effects. If there are any, aside from the improved outlook that follows relief of a headache, they are not known. Of course, a really large dose will produce serious symptoms, including death, but within the range of reasonable doses little if any effect on performance has been noted.

An important methodological point may be mentioned in connection with the study of aspirin, or any other drug, such as morphine, which has definite physiological side effects. Sometimes it is not sufficiently realized that an experienced subject, and sometimes an inexperienced one, knows quite well when the drug has been administered, even when the experimenter tries to control the effects of suggestion by administration of a **placebo.** This difficulty of control is a plaguing problem which is extremely difficult of solution.

Opium and Its Derivatives. Opium is an extract of the opium poppy and contains several physiologically active alkaloids. The most important of these is morphine, to which most of the physiological action of opium is to be attributed. Although morphine has somewhat less violent after-effects, the actions of opium and morphine are very much alike. In both cases, we may generalize by saying that they are narcotic; that is, they produce depressions of the CNS. Opium dulls pain and induces sleep; it may excite for a time, as may all narcotics, before **narcosis** occurs; it reduces bodily secretions, except those of the skin; and large doses depress respiration, to the stopping point if sufficiently large. Morphine is quite similar in action, but is not as likely to be followed by headache and nausea.

Socially, the important aspect of opium and morphine is that they are what is called "habit-forming," although addiction is by no means simply habitual — it is physiological. After either opium or morphine has been taken for a time — ordinarily by smoking in the case of opium, and by injection in the case of morphine — the equilibrium of the body is shifted so that there are acute withdrawal symptoms. These symptoms are so preoccupying that the addict is primarily motivated by them and is much less susceptible to social control than the normal person. Many of the withdrawal symptoms have been produced in monkeys after first "addicting" them to morphine. These behavioral symptoms include much itching of the skin and considerable yawning. In other words, once the bodily economy has been accustomed to opium or morphine there is a new equilibrium established whose upset is even more profoundly motivating than other types of deprivation.

Cocaine. Cocaine is, like opium, a plant derivative, but of quite different physiological action. When cocaine is sniffed the result is stimulation of the CNS, with accompanying excitement and feeling of euphoria. Applied locally to a mucous surface it causes blanching and analgesia. In some responses, cocaine strengthens the action of adrenalin, for example, in the dilation of the pupil. Taken to excess, cocaine can result in psychotic symptoms of great excitement and peculiar sensations of crawling on the skin which are referred to as cocaine "bugs."

Like opium and its derivatives, cocaine is highly habit-forming. Not only is deprivation extremely motivating, but also the addict under the influence of cocaine may become very excitable and unpredictable. In small doses in habitual users, the primary effect seems to be one of dulling of discomfort and probably also dulling of ambition. Cocaine was taken by the nobles and later the people of the old Inca empire in the form of coca, the leaves of which are chewed to obtain the cocaine and other drugs contained therein. A large portion of the population of the Peruvian Andes is to this day addicted to coca. Apparently they are thus made more able to endure the demands of a life of hard toil in a difficult environment.

Benzedrine Sulfate. Benzedrine is a trade name for amphetamine, ordinarily taken internally in the form of the solid sulfate. The effect on most people of a small dose is an increase in alertness and quickness of reaction. It may be used to allay the effects of sleepiness in times of emergency. It is quite similar in its effect to adrenalin. Use of the drug is less widespread than when it was first introduced, because of increasing control of its sale. Some persons have become addicted to the use of inhalers, however, which provide a readily obtainable source of this drug. In contrast to what college students of 10 years ago may have thought, benzedrine is by no means without toxic effects. Leaving aside the danger always inherent in overstimulation of the organism, there is some evidence of auditory nerve degeneration from the prolonged use of benzedrine. The cramming student burning the midnight oil in preparation for an examination is well advised to avoid benzedrine.

Barbiturates. The various barbiturates, all derivatives of barbituric acid, are similar in their action. They are higher CNS inhibitors and, in therapeutic doses, do not adversely affect the vital organs. Very large doses lead to a period of delirium, followed by coma and death. Barbital (one trade name is veronal) is especially powerful and continued use results in habituation. Since the barbiturates affect the higher CNS structures, it can be seen that habitual or unsupervised use is not to be recommended.

ANOXIA

Anoxia, strictly speaking, means "no oxygen." Ordinarily, however, it is used to designate a general inadequate supply of oxygen to the organism as a whole, as contrasted to the inadequate supply to some bodily part. Anoxia can arise from several sources, including lack of absorbing power of the lungs, as in pneumonia, lack of oxygen carrying capacity of the blood (anemia), or low oxygen content of the air in which the organism finds itself. Since the proper functioning of the CNS, and especially the cerebral cortex, depends so directly on the availability of oxygen, the psychological results of anoxia are prompt and extensive.

Before describing some of the more important effects of anoxia, we should consider a general principle which has been alluded to before but which needs reemphasis here. This principle is related to encephalization, and is that the newer, higher CNS structures are more susceptible to oxygen lack. If the oxygen supply is restricted, first the cerebral cortex, then progressively lower structures of the CNS are affected, the spinal cord losing function last of all. It will be noted that this is the same order in which the CNS is affected by alcohol. Oxygen lack will affect more complex psychological functions first, therefore, and just as a person's judgment of his own condition of intoxication is not a good index of his actual impairment, so an aviator flying at high altitude, for example, cannot be trusted to decide for himself his need of additional oxygen. Making such a decision involves the efficient functioning of the cerebral cortex.

Anoxia from Low Oxygen Environments. A low oxygen environment may arise from the depletion of oxygen in the air, as in a submarine, or from high altitude, as in mountain climbing or aviation. The latter situation has occasioned the most research interest and we shall confine our attention to it. First of all, it is necessary to distinguish between the effects of pressure reduction in general and oxygen lack in particular. As altitude increases, the density of the air decreases, that is, there are fewer and fewer molecules per unit volume. This reduction takes place not only for oxygen, which accounts for about 20 percent of the air, but for the other gases, of which nitrogen is almost the entire remaining 80 percent. Since the absorption of oxygen depends upon the number of its molecules per unit volume, not its percentage relationship to other gases, it is possible to reach so high an altitude that even pure oxygen is too rare to provide sufficient molecules. A convenient way of visualizing this point is to plot the total pressure of the air against altitude, as has been done in Figure 23.4. The air pressure, and therefore the available oxygen, has decreased to one half the sea-level value at about 17,000 feet. At this altitude, breathing air enriched with oxygen to double the sea-level oxygen percentage will maintain normal blood oxygen. But at slightly more than 30,000 feet, blood oxygen will drop even if pure oxygen is breathed. A better solution to the problem is to increase the total air pressure, as is done in pressurized airplane cabins.

In the above paragraph we have considered the effect of lowered pressure on the availability of oxygen. Lowered pressure also has other effects, particularly when the pressure is lowered rather suddenly. Most important, lowered pressure may result in what is called the "*bends.*" Bends are severe pains, especially in and around joints, and other symptoms resulting from the presence of nitrogen bubbles in the tissue fluids and blood. These bubbles form because nitrogen, in common with other gases, is not as soluble in water at lower pressures. When the pressure on the body is lowered, the nitrogen comes out of solution in the body fluids. Since nitrogen is not metabolized and must be expired through the lungs, the only cure is to recompress the patient and lower the pressure gradually to the desired level. In our further discussion we shall confine our attention to the oxygen availability aspect of altitude, since virtually all the symptoms of high altitude result from anoxia.

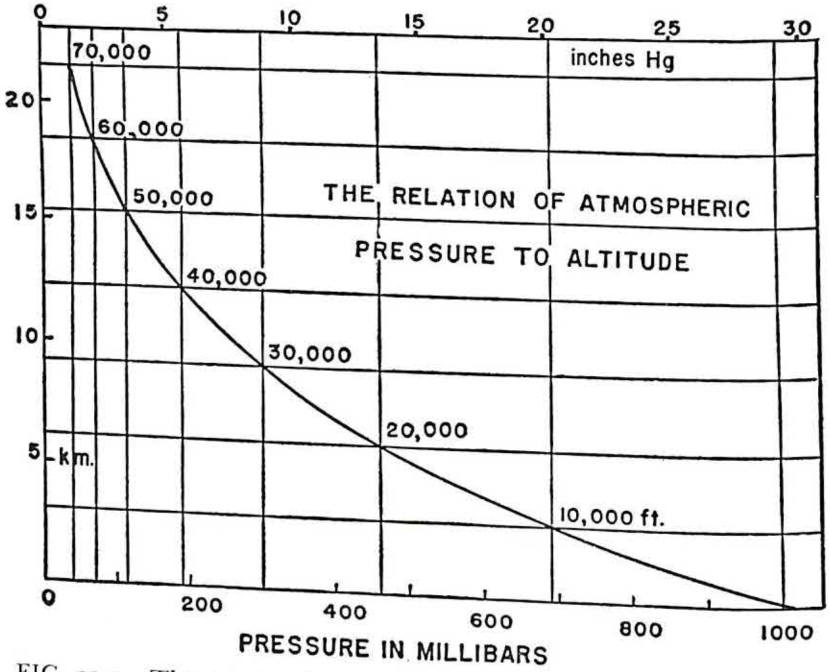

FIG. 23.4. The relation between altitude and average air pressure. A millibar = 1/1000 of a bar; a bar is average sea level air pressure (about 15 lbs. per in.^{2}). (From Bartley and Chute.[11])

Symptoms of slight anoxia may arise even at fairly low altitudes if ascent has been rapid, and especially if the individual is exercising and thus in need of considerable oxygen. A person accustomed to sea-level conditions may have considerable shortness of breath when playing tennis at 5,000 feet. With moderate anoxia one tends to feel uncomfortable, has an irregular pulse, sleeps badly, may become quite irritable, and lacks the ability for sustained exertion. Some persons suffer from "**mountain sickness**" even at elevations below 10,000 feet. The symptoms include nausea, headache, and lack of energy. With prolonged stays at these relatively low altitudes considerable acclimatization occurs, especially as concerns one's feeling of well-being.

Really high altitude leads to impaired function in any measureable sphere, although individual differences are fairly large. Mood changes are observable, with some persons becoming gay and lightheaded, others becoming irritable, much as different persons react to intoxication differently. Considerable acclimatization is possible, at least for selected groups of people. Workers in some of the tin mines of the Andes live at 17,500 feet, and work the mines at 20,000 feet. No doubt considerable selection has occurred through the survival of the fitter persons, but in any event the adjustment is remarkable. The modern era of high-altitude flying has emphasized the importance of a proper level of oxygen for efficient performance, especially since time for acclimatization is not available. The pressurized cabin has done much to improve the safety of aircraft operation.

The EEG reflects the influence of anoxia with great sensitivity.[s] As the oxygen supply is diminished the alpha rhythm becomes slower, then disappears and is replaced by the delta rhythm, which gradually declines in amplitude until the potentials disappear completely. Such a profound effect will occur only with very low oxygen supplies. Even at this point, readmission of oxygen to the lungs results in recovery of brain activity, but the cortex is so sensitive to oxygen lack that permanent damage may be done in a matter of a few minutes. If the electrical potentials of various levels of the CNS are recorded during complete anoxia, it will be found that the activity of the higher centers disappears before that of the lower centers, a fact confirming our earlier generalization about the effect of anoxia on the CNS.

Anoxia from Organic Causes. Anoxia may result from anemia, from failure of normal circulation, as in arteriosclerosis ("hardening" of the arteries), and from other organic causes. The effects are basically the same as those from chronic environmental oxygen lack. Efficiency is lost in a degree determined by the severity of the disorder. In extreme cases, genuinely psychotic symptoms appear. We shall have more to say about the involvement of brain oxidation in psychosis in Chapter 24.

OTHER EXTREME NEED STATES

We have had occasion in Chapter 20 to mention the problem of thirst and have mentioned the importance of water balance to the efficiency and personality of the individual. In man, this is especially important because thirst is not a perfect indicator of actual need. On the desert, voluntary drinking is often necessary to maintain a proper water balance. The symptoms of mild water lack are mostly tiredness, depression, sleepiness, and occasionally hunger. With greater lack, exhaustion will occur, with symptoms of vague discomfort, inclination to sit or lie down, muscular impairment, a rising pulse rate, and a rise in body temperature. These profound symptoms appear before water loss reaches 10 percent of the body weight. It is doubtful that a person can survive a loss of 20 percent of body weight from dehydration. For a 200-pound football player, the loss of 20 pounds (10 percent) in a game would put him very close to the danger point. As far as simple psychological processes are concerned, dehydration, up to a fairly severe degree, has little or no effect. The processes studied include dark adaptation, visual acuity, and manual coordination. Despite these results, the over-all efficiency of the person is reduced, because of the more general changes in alertness and attitude.

Also of importance is the salt (NaCl) loss which is concomitant with the loss of water through sweating. If the lost salt is not replaced, symptoms of exhaustion may appear even though the water intake is adequate. The provision of salt tablets near the drinking fountains in many industries is a recognition of this problem.

For efficient physiological activity, the internal temperature of the human body must be maintained at about 37° C (98.6° F). The metabolic processes of the person, especially the contraction of the striate muscles, are the source of a fair amount of heat. The heat production of a resting wakeful person is roughly equivalent to that from a 100-watt electric light bulb. If the person is climbing stairs, the heat is equivalent to over 1200 watts. For the maintenance of a constant internal temperature, this heat must be lost to the environment of the same rate that it is produced. If heat loss does not balance heat production, bodily temperature rises and death can ensue. Obviously one can tolerate a more extreme environment at rest than when exercising. Many other factors are involved, however, in determining whether or not homeostasis can be maintained and these have been the objects of considerable research by environmental physiologists. Surprisingly high, dry air temperatures can be tolerated for brief periods of time, since the evaporation of sweat is the most important mechanism of heat loss. The highest temperature so far studied is 240° F, which is tolerable for perhaps 20 minutes. The psychological effects of high heat loads depend upon whether short or long-term testing is done. Even under extreme conditions it is difficult to demonstrate much decrement in ability for brief tasks.[9] On a longer term basis, however, the inclination to do work, mental work included, diminishes, even though ability may not show much reduction if motivation is sufficient. If conditions are not too extreme, some acclimatization occurs — a process accelerated by exercise.

When skin temperature falls, hypothalamic reflexes constrict the blood vessels of the skin and may induce shivering. Actually, the task of combatting real cold is beyond the physiological mechanisms of the body and considerable research has been directed at the design of proper protective clothing — research which has not resulted in any improvement on traditional Eskimo winter dress. Aside from survival, the problem of cold is that it leads to numbness and stiffness, especially of the hands, and manipulation of tools and instruments becomes very difficult. After a time in the cold, internal bodily temperature drops and the person drifts off into unconsciousness. Until this point, the person may be uncomfortable and be distracted by his discomfort, but actually loss of ability is not demonstrable.

Another extreme need state is represented by prolonged or severe pain. Even relatively mild pain can influence the patterns of reflex and voluntary movement. For example, handwriting is altered by pain in the hand and a sore toe makes limping very hard to prevent. As pain becomes more severe, it also becomes more preoccupying and more distracting. Autonomic reflexes are initiated and behavior will be interfered with, even to the extent of fainting. Prolonged pain, even mild, can leave its mark etched in the face of the sufferer. Other bodily changes which can result from prolonged pain have been mentioned in Chapter 8.

Upset hormone function represents our last type of extreme need state. Considerable discussion of hormonal influences on behavior and personality has been given in Chapters 15 and 20. Suffice it to emphasize here, that departure from an optimal hormonal balance represents a somatopsychic influence which may alter behavior profoundly.

Suggested Readings

1. Bartley, S. H., and Chute, Eloise, *Fatigue and Impairment in Man.* New York: McGraw-Hill, 1947.
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24 Somatopsychological Problems: continued

In the preceding chapter we have considered a number of problems concerning the effects of bodily factors on behavior. Our concern now is with behavior which is frankly abnormal, especially psychotic behavior. Our interest with this topic stems from two main sources: first, the tremendous social importance of mental disorder; and, second, the increasing interest in physiological research related to its causation and treatment. The social importance of mental disorder can be summarized very briefly. There are at present more than one half million patients confined in state and federal mental institutions in the United States. This means that there are more hospital beds occupied by the mentally ill than by those suffering from all other disorders combined. Of the half million mental patients, almost half are diagnosed as schizophrenic, and so the social importance of discovering the cause and treatment of this disorder alone can scarcely be overemphasized. The growing body of physiological research, still exploratory and tentative in nature, is an expression both of this social importance and of a suspicion that such a profound disorder involves more than extreme maladjustment. Whether or not this suspicion will be justified cannot at present be definitely decided. This chapter will be devoted largely to a consideration of the present status of the evidence.

Since our concern is with psychotic behavior, it is well to have in mind what the term psychotic implies. For our purposes, we may say that a psychosis is a disturbance of behavior of such severity that the individual cannot adjust to the day-to-day demands of a normal environment to the extent that he must be kept under special care for the protection of himself and society. Psychotic symptoms may include loss of contact with the immediate environment, delusions, and disturbances of emotional behavior and of consciousness. Sometimes behavior may be most bizarre, while in other cases a superficial impression of normality may be evident. Unfortunately, no really good classification of psychoses exists and it is likely that proper categorization will arise only after the causation of different types of abnormality has been worked out. In any event, we shall confine our discussion to the more serious and profound disorders, since there is less adequate evidence that the less severe disorders (the "neuroses") have a primary physiological basis, although various factors influencing the physical well-being of the person may be of secondary importance.

Toxic Agents. There are a large number of specific factors which are known to produce mental disorders and a brief mention of these may help to show the sort of symptoms which are characteristic of severe disturbance of cerebral function. There are many toxic substances used in modern industrial processes which produce well-defined psychoses, although the importance of any one of these fluctuates as commercial production techniques change. At the present time, the toxic substances of greatest numerical importance are lead, mercury, manganese, carbon disulphide, and carbon monoxide. Lead poisoning is not uncommon and may lead either to progressive mental deterioration or to delirious episodes with confusion, outbursts of violence, visual hallucinations, delusions of persecution, and the like. Mercury poisoning may produce irritability, timidity, discouragement, and sometimes emotional outbursts. Manganese poisoning involves largely the extrapyramidal motor systems and the basal ganglia, with tremors and gait and speech disturbances; but it also may involve mental symptoms of restlessness, elation, and uncontrolled laughing or crying. Carbon disulphide (used in rayon manufacture) causes all kinds of neurological symptoms, and also fatigue, listlessness, insomnia, loss of memory and uncontrolled anger. Carbon monoxide poisoning, which occurs sometimes by chance through faulty functioning of motors or heating devices, and sometimes by design in attempted suicides, produces confusion, delirium, and apathy. It may produce a temporary or even permanent deficit of an intellectual nature due to anoxemia of the brain resulting from the combination of the hemoglobin of the blood with the carbon monoxide, to the exclusion of oxygen.

Drugs. Drugs are another source of psychoses and are responsible for about 1 percent of all admissions to mental hospitals. Of these, the bromides and barbiturates are the most important. Bromide poisoning is fairly common (it can even be produced by a readily available commercial drug preparation if taken in sufficient quantity), and is particularly important in elderly people and in arteriosclerotics where the tolerance is low. The prominent symptoms are delirium, fear or depression, and paranoid ideas. The barbiturates, on the other hand, render the victim drowsy, disoriented, and euphoric in a silly way. Opium and its derivatives rarely produce psychoses, although there are temporary psychotic symptoms, and marihuana probably never. Cocaine addiction, however, may produce delusions of persecution, terrifying visual hallucinations, and disorders of somesthetic perception resulting in the feeling of insects crawling around under the skin. There are a few other drugs which also produce psychoses, but they are very much less common. Chronic alcoholism of long standing may eventuate in psychoses, among them delirium tremens and Korsakoff's syndrome. The primary difficulty, however, stems from nutritional deficiencies, particularly vitamin B deficiency, and general debility. In Korsakoff's psychosis, there is amnesia, relation of fictitious memories, a kind of jovial air, and utter dependence upon immediate observation. Sometimes recovery occurs, but sometimes the degeneration in the nervous system has gone so far as to be irreversible; and although some improvement occurs over a course of a few months, there is permanent impairment of memory, judgment, appropriate emotional response, and esthetic awareness. In addition to these well-known drugs, there are a number which have been used in experiments to produce psychotic symptoms in both man and lower animals.

Injury. Another obvious source of severe mental impairment is injury to the brain. Brain injuries may arise from blows of various sorts to the head. Simple concussion, in which there is disordering of neuronal functioning due to mechanical jarring of the neurons, may lead to psychotic symptoms. Where there is also hemorrhage, the results are likely to be more serious and the exact symptoms shown will, of course, depend upon the location of the rupture. Direct damage to the brain structure, as by a gunshot wound, may also cause psychotic symptoms, again depending upon the location of the injury. Tumors may cause similar symptoms. Such traumata often cause epilepsy (discussed in greater detail below) and produce obvious changes in the EEG in the region of the damage. The disorder known as " punch drunk," which occurs in boxers who have absorbed too many blows, is a direct result of the frequent mechanical jarring of the brain, every knock-out blow probably destroying some neurons. There is deterioration of muscular skill, of intellect, and of personality, the person frequently appearing a little drunk, often talkative and gay, but with poor attention and memory.

Diseases with Psychotic Symptoms. Various disease processes may produce serious and diverse psychological symptoms. Some of these are temporary, as with the delirium that accompanies a high fever.

In some diseases, the psychotic symptoms are among the most prominent aspects and are of longer duration. In severe cases, chronic residual damage often is left even after the acute phase has passed. Among these infectious diseases we shall mention but four of the most dramatic. The first of these is general paresis, caused by syphilis of long standing which attacks the nervous system. The cerebral symptoms usually develop 5 to 30 years after the primary infection, and are due to atrophy of the cortex, particularly the frontal lobes. The mental symptoms include fatigability, irritability, marked unconcern about social amenities, lack of consideration for others, deterioration in professional skills, poor judgment, inability to see the consequence of errors, apathy, depression, and eventually delusions. This disease is less common now than formerly, but does occur with some frequency. It is the subject of Ibsen's play, *Ghosts*. If primary syphilis is properly treated, general paresis never develops; modern routine prophylaxis, such as practiced by the Armed Forces, would totally prevent it.

Epidemic encephalitis, sometimes called sleeping sickness, is not at present a very serious threat in this country, but it has been in the past and still is in some other parts of the world. It is due to a virus. There are two acute forms, not just the one which occasioned the popular name. One form, the " lethargic," produces stupor, slowing of all intellectual activity, aphasia, and symptoms of brain-stem involvement. The patient may sleep for weeks and, if aroused, promptly falls asleep again. Reference to the discussion of sleep and its physiological basis will throw light on this symptom; damage to the brain-stem reticular formation and the arousal mechanism would be expected to result in symptoms of this sort. The other " acute " form of the disease apparently spares this region. The symptoms are almost opposite, being characterized by restlessness, irritability, insomnia, and convulsive seizures and other disorders of movement. In the chronic form of this disease, after the acute phase has passed, the most prominent symptoms are movement disorders of various kinds, emotional changes such as mania or depression, and lack of concern for the results of behavior. Only in young children is there any permanent impairment of intellectual ability; it is accompanied by behavior disorders of a sort which render their care at home extremely difficult. They are " naughty," disobedient, stubborn, and frequently delinquent. Usual methods of behavioral control do not produce improvement.

Epidemic cerebrospinal meningitis in its acute phases produces delirium, confusion, and restlessness. It is due to inflammation of the meninges. There may be permanent impairment of memory, various types of paralysis, inability to concentrate, moroseness, and irritability.

Finally, rheumatic fever or any severe fever may cause an encephalitis, although it by no means always does. When this occurs there may be permanent motor disorders with jerky, involuntary movements, incoordination, and grimaces. Marked emotional instability is associated with this and there may be behavior disturbances of the sort mentioned above which are very difficult to deal with. A child with this disorder, although contrite after such behavior, seems unable to do anything to prevent it under the normal home conditions.

Table 24-1. Distribution of Psychoses among Parents and Siblings of Psychotics*

Type of Psychosis in Patient	*Expectancy Rate in General Population*	*Expectancy Rate for Family Members*	
		Schizophrenia	*Manic-Depressive*
Schizophrenia	0.9	9·7	0.1
Manic-Depressive	0.4	—	21.2

* Adapted from Kallman, F. J.[1]

Genetic Factors. Recent investigations of the genetic factors in psychoses provide interesting evidence of the existence of important biological influences in spite of the unsatisfactory nature of the classification of psychoses. Analysis of the distribution of psychosis reveals much higher rates among relatives of psychotic patients than in the general population for the particular general class of psychoses which the patient has, but not for others. Table 24-1 shows some of these results for the two major classes of "**functional**" **psychoses.**[1] For **schizophrenia,** the expectancy rate in the general population is only 0.9, but for the close relative of a schizophrenic patient it is 9.7. The expectancy rate for **manic-depressive psychosis** is only 0.4 in the general population, but is 21.2 for relatives of a manic-depressive, and 0.1 for relatives of a schizophrenic. Note that the relatives of patients with one type of psychosis virtually never develop the other psychosis. Although these figures, based on very large numbers of cases, lend some credence to the hypothesis that there are important biological factors in psychoses, much stronger evidence is presented in Table 24-2,[1] where expectancy rates for various degrees of closeness of blood relationship are presented. As the closeness of blood relationship increases, the expectancy rate goes up. For half siblings, the rate is half that for full siblings; it is no different for full siblings than it is for

Table 24-2. Distribution of Psychoses according to Closeness of Blood Relationship to Psychotic Patients*

		Expectancy in Families of Psychotics				
Type of Psychosis	*Expectancy in General Population*	*Parents*	*Half Siblings*	*Full Siblings*	*Dizygotic Cotwins*	*Monozygotic Cotwins*
Schizophrenia	0.9	9.3	7.1	14.2	14.5	86.2
Manic-Depressive	0.4	23.4	16.7	23.0	26.3	95.7

* Adapted from Kallman, F. J.[1]

nonidentical twins, nor should it be, because nonidentical twins are no more closely related genetically than are siblings born at different times. But the rate for identical twins, having identical heredity, is amazingly high. It can hardly be doubted that a powerful genetic factor is at work here. How these genetically controlled factors operate and what relationship they bear to other physiological factors present in the individual and to possible precipitating environmental factors is not known as yet.

Structural Brain Changes. One obvious approach to the discovery of causes of psychotic behavior is to examine the brains of psychotic individuals. The examination may be gross or may involve histological techniques; i.e., the slicing and staining of brain tissue and subsequent examination under a microscope. In some cases, such as those diagnosed as tumor, arteriosclerosis, or rheumatic brain disease, gross changes are present in the brain which adequately account for the deviant behavior. In many cases, however, no such gross pathology is present. Histological findings have proved conflicting. In the case of schizophrenic, manic-depressive, and other "functional" psychoses, all the positive findings have been challenged by competent histologists. The difficulty is partly a matter of the normal variations to be expected in tissues, partly due to changes induced by the handling of the tissue which may mask more subtle changes associated with the disorder, and also partly because the nervous system changes associated with psychotic behavior may be at the molecular level and hence beyond the realm of present histological techniques. At present very little psychotic behavior can be directly associated with brain changes, although the possibility of discovering such relationships in the future should not be discounted.

Endocrine Disturbances. Disorders of the endocrine glands have been discussed previously, but they must be mentioned here because some of them may result in psychoses when the disturbance is severe. Insufficient anterior pituitary secretion may lead to a vegetable-like apathy, which is relieved by the administration of thyroid extract, testosterone, and cortisone together. The extreme forms of thyroid disorder may lead to psychosis. Myxedema produces delusions and hallucinations in nearly half the cases and, also, may result in either mania or **melancholia.** In hyperthyroidism, the typical irritability, euphoria, and **hypomania** sometimes lead to real psychosis.

Disease of the Islands of Langerhans causes excessive secretion of insulin which produces hypoglycemia, with the usual depression of cerebral functioning and severe behavioral changes. Deficient secretion of gonodal hormones, on the other hand, apparently does not produce psychotic symptoms, but an alteration in gonadal hormone balance may do so. **Involutional melancholia,** a psychotic depression which may occur at menopause, has long been considered due to deficiency or imbalance of hormones. There are some cases in which hormone therapy is very effective in relieving the symptoms. In other cases, it is likely that the depression was not caused by hormonal imbalance, but by some unknown factor which may operate in melancholia occurring at any time. Therapy with all the hormones discussed above has been tried on schizophrenics with little effect. Hormone therapy cannot be expected to be effective when the basic physiological disorder is not hormonal.

Recently there has been a considerable flurry of excitement concerning the possible role of the adrenal cortical steroids in schizophrenia. It has long been known, of course, that marked deficiency of adrenal cortical secretion results in Addison's disease, a condition characterized by muscular weakness, apathy, and paranoid attitudes, sometimes with hallucinations. Our concern is not with such obvious abnormalities but with the response of the adrenal cortex to stress, the "general adaptation syndrome" described in Chapter 15. One important factor enabling the organism to withstand stress is the increased production of cortical steroids. The response occurs after stress produced by such divers things as trauma, hemorrhage, burns, electric shock, extreme heat or cold, exposure to x-rays and solar radiation, and emotional conflict. The condition known as "shock" may result and recovery from shock is presumably due to the normal stress response. The stress response has been examined in connection with schizophrenia because of the fact that so far the causative factors in this psychosis have escaped detection, and because of the lack of contact of the schizophrenic with his environment and his inappropriate emotional response. There are difficulties in this research because of the fact that the indicators of adrenal cortical response do not agree perfectly, and because of the looseness of the definition of stress. Results have therefore been conflicting. In one recent study, where 100 schizophrenics were compared to a normal control group under several kinds of experimental stress, it was found that there was no real difference in the baseline level of adrenal cortical response indicators, but that the schizophrenics did not show an increase in their output of adrenal cortical hormones under stress, whereas the normals did.[2] Others have not found as clear-cut results. One difficulty here is that the diagnostic category, schizophrenia, is probably not a good one, since it is based entirely upon psychological symptoms. There may actually be a number of different disorders involved, requiring different treatments. In general, hormone therapy with ACTH and cortisone has not proved beneficial. But this is an active research field and it is too soon to draw any definite conclusions.

Metabolic Disorders. Metabolic disorders of several sorts have been investigated, among them the oxidative functions in the brain, specific enzyme systems, and protein metabolism. There are two obvious and important factors in the oxidative functions of the brain contributing to the cerebral metabolic rate, the availability of oxygen, and the availability of glucose, the only important food-stuff of the CNS. It has already been pointed out in Chapters 19 and 23 that the metabolic rate of the brain is high — 20 percent of the total basal metabolic rate. This is a reflection of the oxygen consumption of the brain and emphasizes, even more than the EEG records, the perpetual activity of the brain. The brain is also especially susceptible to oxygen deprivation and the most susceptible structures are those most recently evolved. Thus, the cerebral cortex is damaged first, together with higher-order psychological functions in a very few minutes, whereas the breathing centers in the medulla survive much longer. The blood supply is greater to the cortex than to cerebral white matter, and greater to those regions which are most active than to those which are not as active. In view of the special sensitivity of the brain to oxygen lack, it is reasonable to assume that some interference with oxidative function might be the basis for some mental disorders. Unfortunately, this is not an easy hypothesis to investigate. It has been shown that the cerebral metabolic rate is reduced in general paresis and after lobotomy, but this might be expected because of the destruction of nerve cells. Some therapies which are used for paresis (e.g., fever) and for cretinism (thyroid extract) raise the cerebral metabolic rate, but they also accelerate enzyme activity. Investigations of the cerebral metabolic rate in schizophrenics and in depressive psychotics have usually shown it to be normal, but recent evidence has thrown some doubt upon this finding.

An interesting aspect of this topic is the role of asphyxia at birth in the etiology of mental deficiency, mental disorder, and perceptual and motor deficits. Because of the facts that asphyxia at birth causes one-fourth of fetal and infant deaths and that many of those who survive have obvious damage to the CNS, as in the case of spastics, it has occurred to one investigator that probably many other survivors have less obvious, but none-the-less real damage. He designed a study to test the hypothesis experimentally, using guinea pigs rather than human beings.[3] One guinea pig fetus was delivered by Caesarian section, and its litter-mate control was asphyxiated by cutting off maternal circulation to the placenta for varying periods of time, from 4½ to 23 minutes — about the limit of survival of a guinea pig fetus without oxygen — and then revived. Without exception all the asphyxiated animals showed some symptoms of nervous system damage, from coma to various kinds of motor and perceptual deficit. Nearly every animal asphyxiated for more than 8 minutes showed structural damage to the brain, and nearly all of this group were inferior to their litter controls in maze learning. A good deal of support is thus lent to the original hypothesis, and the suspicion is strengthened that difficult births or ones involving any prolonged asphyxia in human fetuses may cause cerebral disorder.

The role of the availability of glucose in cerebral metabolism appears to be less critical because of the protective homeostatic mechanisms which ensure the brain's glucose supply at the expense of other tissues. However, a significant and prolonged fall in blood sugar, which can occur in both natural and artificial circumstances, profoundly affects the functioning of the brain. Extreme hypoglycemia can be tolerated by the brain for only a few hours without irreversible damage. Such hypoglycemia may occur in various diseases, in some kinds of poisoning, or artificially in insulin shock therapy often given for " functional " psychoses. The symptoms are likely to be hallucinations, amnesia, outbursts of anger, and in extreme cases, coma.

An important aspect of metabolic disorder of the brain involves enzymes. This work is extremely tentative as yet and only a few enzymes have been investigated. Research on enzymes is made significant, however, by its concern with very subtle changes, in contrast to the grosser changes we have been considering, and because enzyme changes are known to be of importance in the disorders of other organs. Cholinesterase in excess of normal has been found in cortical tissue from the frontal lobes of some deteriorated schizophrenics.[4] This has led to some speculation that the underlying pathology is a chronic excessive discharge of neurons in the frontal lobes with subsequent exhaustion. In the same specimens, another enzyme, acid phosphatase, appeared to have a somewhat reduced reaction in cortical cells. A more thorough study[5] of still another enzyme, carbonic anhydrase, revealed patterns of distribution within the nervous system, in both animals and men, which appeared to be related to CNS function. There were species differences found and, also, differences at various stages of development of the nervous system. The amount of carbonic anahydrase was found to increase with higher levels of the brain in both gray and white matter. Within the cortex itself, differences were found for various functional areas. This enzyme is not present at all in the cerebrum until late in fetal development in lower animals and there is none in the human cerebrum at birth. Such findings relate well to behavioral development. Analysis of enzyme content of brains of psychotics of various diagnoses showed a regular increase as higher levels of the nervous system were involved, but there was a disturbance of the pattern of distribution. Analysis of 105 brains of mental patients showed that many had an enzyme content in the prefrontal areas which was less than the normal amount.

A specific metabolic disturbance which has been investigated in connection with mental disorder is that of protein metabolism.[6] In a small sample it was found that the protein-producing system of the frontal cortex was poorly developed in psychotics. Injection of a substance which was shown to produce increases in CNS protein content in rabbits resulted in some improvement in chronic schizophrenics and other psychotics, though some of the improvement may have been spontaneous.

The Electroencephalogram. EEG is an important aspect of research upon the function of the brain, but it has been of little aid to the psychiatrist or clinical psychologist as a diagnostic tool except in certain specific disorders, notably epilepsy and brain tumors. Marked disturbances of the normal pattern occur in both these conditions. Slow rhythms are usually found in the region of tumors. In epilepsy, there is a typical slow wave and spike pattern, and spikes typically are associated with the actual seizures. Slow rhythms (below 4 per second) without spikes are seen whenever cortical function is depressed, as in hypoglycemia, anoxia, anesthesia, sleep, and normally in infancy. The EEG is not of assistance at the present time in diagnosing schizophrenia or other functional psychoses.

The Autonomic Nervous System. The role of the ANS in mental disorder has been studied infrequently and then largely in terms of blood pressure responses. In older patients with a long history of psychosis, the ANS seems to be less reactive than in normal individuals. In a recent study of 110 cooperative schizophrenics with a recent onset of the disorder, it was found that they show a marked apparent hyperactivity of the sympathetic system.[7] In 98 psychoneurotics, the apparent dominance of the sympathetic system was less extreme, but was significantly different from normal.[8] Other lines of evidence suggest that schizophrenics and psychoneurotics do not necessarily show the same patterns of autonomic function, and the different patterns may be indicative of fundamentally different mental characteristics in the disorders. This work is still exploratory in nature.

Aging. Finally, the process of aging produces physiological changes which are of significance in mental disease. It is a matter of some debate whether these changes are natural and inevitable processes or whether they are basically the result of disease processes, dietary imbalances, or metabolic disorders. In any event, at the present time these changes appear to be unavoidable. In many cases they do not lead to real disorder, but they may. The two major sources of physiological difficulty are a deterioration and atrophy of the brain cells which is not particularly selective and leads to senile psychosis and arteriosclerosis of the brain (hardening of the cerebral arteries). The latter condition may affect certain areas more than others and, therefore, lead to more varied and less general symptoms. It results in impoverishment of circulation in an area with resultant degenerative changes in the brain cells.

Mental Deficiency. Following the above discussion of specific physiological factors in mental disease, some consideration of the disorders themselves is in order. Mental deficiency is a real social problem, and is of interest here because of the large number of physiological causes which produce it. There are a number of types of mental deficiency which are inherited, including mongolism, microcephaly, and a condition called phenylpyruvic amentia. These conditions are usually recessive, genetically, and some are sex-linked. The RH factor accounts for some cases of feeblemindedness, and is caused by a combination of an inherited characteristic plus the interaction of this characteristic with the maternal environment. How these inherited characteristics produce their effect is not known.

Another group of causes for mental deficiency operate during fetal development: nutritional deficiencies of the mother (which may be either metabolic disorders or inadequacy of diet), toxins, X-rays, or infections during pregnancy. Birth injuries of several sorts account for a large number of cases: mechanical damage to the brain, hemorrhage, or asphyxia. In a study of 900 children whose brain defects were thought to be related to conditions occurring at birth, 70 percent had a history of asphyxia.[9] Hydrocephalus, i.e., water on the brain, due to abnormal increase in cerebrospinal fluid and consequent damage to brain cells by increased pressure, is another cause for mental deficiency. Premature closing of the skull also puts pressure on the developing brain and damage to neurons results. Cretinism, produced either by thyroid deficiency itself or by disease of the anterior pituitary and, thus, deficiency of the thyrotrophic hormone, produces feeblemindedness which can be prevented if treated in time. Specific metabolic disorders may also cause mental deficiency. Recently it has been shown that feeblemindedness due to phenylketonuria, or poisoning by an amino acid called phenylalanine, can be markedly improved by feeding a diet from which this substance has been extracted chemically.[10] Moreover, various toxic, virus, and traumatic conditions in the early life of the child may also produce mental deficiency. As an example, in a study of 128 children who had whooping cough at a very early age, 15.6 percent became intellectually retarded.[9] The percentage was higher for the first two years of life. Other viruses cause similar deficiencies.

Epilepsy. Epilepsy refers to a group of symptoms which may be produced by a number of conditions, including brain tumor, mechanical injury to the brain, and some unknown physiological changes which cannot be detected by present techniques. It is not a specific disease, but rather a group of convulsive disorders, with some associated symptoms. In the more severe forms, there are convulsive movements, autonomic disturbances, loss of consciousness, and dramatic changes in the EEG pattern. In **grand mal** epilepsy there is an aura, or premonition of the attack, which may consist of various unpleasant hallucinations, ordinarily typical for a given patient. There follows a sudden loss of consciousness, when the patient falls and often injures himself, accompanied by tetanic contraction of skeletal muscles, abnormal autonomic responses, and finally convulsions. Sleep, or coma, usually ensues. The EEG pattern is characterized by very fast, high voltage activity. In **petit mal** epilepsy, the convulsive movements are likely to be limited to twitching of the eyelids or head and the loss of consciousness is brief and usually does not involve falling. Petit mal often develops into grand mal. There are other related convulsive disorders, sometimes called psychomotor epilepsy, in which the involvement is so slight that it may not be detectable except with EEG. In most cases of epilepsy, there is definite abnormality of EEG pattern, even when seizures are not in progress, but about 15 percent of epileptics do not show these abnormalities, and about 10 percent of presumably " normal " individuals do show abnormalities similar to those found in epileptics. Some of these may develop epilepsy later. There are about 650,000 epileptics in this country, most of whom do not require institutional care.

Senility. Senile psychosis is an important diagnostic category, and more and more psychoses of this type appear as life span increases and as the population becomes, on the average, older. Old age does not necessarily bring senility and senility may be relatively mild; but often a real deterioration of brain tissue ensues and psychotic symptoms appear. The brain changes which occur in this disorder involve reduction in the number of neurons. There is loss in brain weight, due to atrophy of cells, and other cells show degenerative changes. The symptoms often start as an intensification of ordinary, nonpathological traits of old age: loss of interest in the environment, rigidity of habit patterns, a tendency to reminisce. The psychosis may involve several of a large number of symptoms. There may be mental deterioration with loss of memory for recent events, loss of contact with the environment, suspicion and resentment of younger family members, abnormal sexual behavior, carelessness of appearance, confusion about present events, sometimes hallucinations and delusions, and many others. The hoarding of wealth or other objects by elderly recluses, often noted in the newspapers, is rather typical of the senile pattern. This psychosis is not the same as that produced by arteriosclerosis of cerebral vessels, although both typically occur in old age. In the latter, the destruction of tissue may be more selective and there is usually some awareness of the condition.

Functional Psychoses. Schizophrenia is an important diagnostic category at the present time and a large percentage of psychotics are so diagnosed. Although we have already indicated the physiological factors which have been considered in relation to schizophrenia, it may be helpful to summarize them together here. Schizophrenia is probably not a single disorder. It is probable that a number of conditions can be responsible for the symptoms, so one should not expect a single factor to account for all cases thrown into this " wastebasket " category. There are a large number of symptoms usually associated with the conditions described as schizophrenia, but the central core without which the case would probably not be so classified are the following: (1) feeling-tone is usually lacking or inappropriate; the patient may show no emotion at events which normally induce strong emotions, or he may giggle where you would expect grief; (2) there is a progressive withdrawal from environmental and social contacts; the patient retires more and more into a solitary existence; (3) attention is concentrated on internal events, and it is difficult to get the individual to attend to external occurrences; (4) associations of ideas are disturbed, bizarre, and illogical, and symbolic systems are developed which are peculiar to the individual; (5) delusions and hallucinations are extremely common. Schizophrenics are usually classified into four types, but the evidence for these types is not good. As already indicated, there is some evidence for the existence of a number of types based upon biochemical, endocrinological, and other physiological factors, but they do not appear to correspond to the present diagnostic classes.

The physiological factors which have so far been studied have proved suggestive, but not conclusive. Histological investigations have not uncovered any gross CNS defects, but the assumption is that the disorder is at the metabolic level and would not show up as gross degeneration. Cerebral metabolic rate has been studied a number of times and the present consensus is that there are no differences between normals and schizophrenics, although there is some disagreement. There is a growing body of evidence that some enzyme systems are deranged in schizophrenia, but it is too early to say how important this will prove to be. The endocrines are apparently not of specific importance, with the possible exception of the adrenal cortex. There is some evidence that cortical steroids are not produced normally in response to stress in schizophrenics, although it is not clear just what could be expected to be stressful to a schizophrenic. Also, protein metabolism may prove to be a factor differentiating schizophrenics from normals, but research is not far advanced in this area. Finally, some differences in ANS balance have been reported, but need verification.

A difficulty which besets all clinical research is the fact that a certain percent of spontaneous remissions must be expected. Any claims for beneficial effects of therapies must take this into account and produce a significantly greater number of "cures." In the case of schizophrenia, the percentage of spontaneous remission varies from 18.2 percent to 65 percent, depending upon the particular study and the duration of the illness at the time of tallying.[11] This makes evaluation of therapies and causes difficult. One finds that the conditions which make for greater therapeutic success (short duration of illness, for example) also make for greatest percentage of spontaneous remissions. Psychotherapy has not been able to overcome this difficulty to the satisfaction of research-minded workers. One line of evidence which lends some confidence to the physiological approach is the discovery of genetic factors in schizophrenia. This direction will probably be pursued with more vigor in the future. So little is known of other psychiatric categories that it is not profitable to discuss them.

Therapies. A brief discussion of some of the currently employed therapies is of interest. Psychotherapy, as already indicated, has not proved outstandingly successful for schizophrenia and other so-called functional psychoses, although it may be a useful adjunct. Organic therapies have not proved outstanding either; but, since they are more specific, the difficulty may be that disorders of very diverse etiology are treated similarly or that the wrong agents have so far been tried. A popular treatment at present is prefrontal lobotomy and related surgical procedures. This involves severing some or all of the prefrontal lobe connections to the rest of the brain. Usually lobotomy is performed when other treatments fail and there is no reasonable hope of recovery. In some cases it appears to make the patient more manageable, and less unhappy. There is argument as to the amount of intellectual deterioration it produces. It has been used in some schizophrenic cases, in extremely agitated cases, and where obsessive behavior patterns dominate.

Endocrine therapies have not proved themselves except in those cases where the primary disorder is a severe endocrine imbalance. The use of cortical steroids for the treatment of schizophrenics is still in the experimental stages, with both positive and negative evidence available. The shock therapies — electroshock and insulin shock — are widely used. Insulin, producing a hypoglycemic coma, is employed less frequently than electroshock. A series of shocks is given the patient, each resulting in coma and usually in convulsions. Presumably, the oxidative functions of the brain are altered by the shock, and what rationale there is behind the therapy explains that the response patterns are broken up.

Recently carbon dioxide inhalation therapy has been introduced. A series of treatments, consisting of inspiring a 30-percent CO_2–70-percent O_2 mixture until consciousness is lost, has been reported of benefit to some kinds of disorders. The rationale behind the therapy is a counteraction of what is presumed to be a hyperexcitability of the hypothalamo-cortical system. In the course of a treatment the patient has various motor and sensory symptoms. A number of other techniques have been tried, more or less on a trial and error basis, and although some successes are always reported there is not as yet any convincing evidence of their efficacy. An example is the use of glutamic acid, at first reported to improve intelligence. Unfortunately glutamic acid will not pass the blood-brain barrier, so any reported effect upon brain function directly would be very much suspect.

In conclusion, we can state that research on the physiological bases of mental abnormality has merely begun. It seems entirely reasonable, however, to suppose that mental abnormality, whether it be styled "organic" or "functional," can have physiological bases. When the physiology of individual differences is better understood we shall better understand the psychology of individual differences.

Suggested Readings

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25 Some Problems in Physiological Psychology

In the first and second chapters the basic point of view of this book was discussed in some detail. Briefly put, this view is that living organisms are psychobiological; that is, they are themselves not naturally or really divided into physiological parts and psychological parts. This point of view implies that understanding behavior involves the consideration of physiology as well as psychology. This is the basic justification for the study of physiological psychology. An understanding of the ways in which the CNS, the ANS, and other bodily mechanisms function enriches our appreciation of the study of behavior.

Our survey of physiological psychology has, of necessity, considered the organism in piecemeal fashion. In the first chapters it was necessary to introduce the fundamental physiological and anatomical facts upon which to base the more psychologically oriented later chapters. Only in the last chapters was it possible to deal with the organism-as-a-whole; the organism as a sensient member of society, depending for its normal adjustment to the world around it upon the orderly functioning of its internal economy. But this is the " real " organism; the analyzed organism of the early chapters is the product of necessity, of the impossibility of comprehending the entire organism at once.

In our survey of facts and principles, it was necessary to express ignorance on many occasions. Many of the most basic problems of physiological psychology remain to be elucidated. Some of these problems are proper subject matter for the biochemist and neurophysiologist and are currently being attacked with considerable energy. Still others will require the combined efforts of physiology and psychology for their solution.

In order to emphasize the range and importance of research effort in the general area of psychobiology, we have outlined in this chapter some of the more important problems. Not all are of equal importance, of course. And new insights and new approaches will arise to open up new vistas. But at the present, the problems given here are valid and of interest.

Some Problems of Nerve and Receptor Cell Action. Although the study of nerve and receptor cells is largely the concern of the physiologist, the outcome of the research is of interest to the psychologist. Bearing particularly on the problem of understanding behavior are the following:

1. *The Nature of Excitation.* In Chapter 6 and in the other chapters dealing with sensation, you read many times the statement, " We do not know the true nature of the adequate stimulus." To this we might have added, " In no case do we know the true nature of excitation in a given receptor or other nerve cell." Of particular interest is the manner in which the differentiated receptor cells are excited. What accounts for the reaction of the rods and cones to light and, among them, to different kinds of light? How explain the reaction of the olfactory membrane to odors, of the taste buds to chemical solutions, of the Organ of Corti to vibrations, or of the differentiated receptor cells of the skin in different forms of energy change? And, perhaps even more fundamental, how explain the reaction of free nerve endings to the group of stimuli we have often mentioned — mechanical, electrical, chemical, and thermal — which serve also to excite the cell membrane of undifferentiated organisms like amoeba?

2. *The Nature of Synaptic Transmission.* A closely related problem is the manner in which one nerve cell activates another. We have alluded briefly to the theories of electrical and chemical transmission of the nerve impulse across the synapse, but the problem remains unresolved. Probably chemical transmission is the rule in the ANS. But do synapses in the CNS work in the same way? The answer will be of importance to the physiological psychologist.

3. *The Nature of Inhibition.* The nature of excitation of a single cell or of one cell by another is only part of the problem of individual nerve cell action. Another important problem is how one nerve cell can inhibit the action of another. We know that such inhibition does occur but the exact manner in which it is brought about is still not completely known. Moreover, it is not certain whether a given nerve cell can have both excitatory and inhibitory functions at different times and under different conditions. Inhibition is as important a factor in our behavior as is excitation. Without it we could not walk or engage in any form of coordinated activity, let alone attend to any given thought. It seems clear, then, that much more must be known

about the basic process of inhibition if we are to have any real understanding of behavior.

4. *The Nature of Receptor Adaptation.* We know that adaptation to stimulation of the sense organs occurs, and the facts of adaptation have been mentioned in the chapters on the different senses. But where does the adaptation occur? In some cases — touch, vision at least in part, for example — the receptor cells themselves adapt. But in other senses, this is not true and adaptation takes place in the CNS. The elucidation of these relationships will be of considerable interest to the psychologist interested in sensation.

Patterns of Nervous Action. Patterns of nervous action may involve any group of two or more cells and, of course, they have temporal characteristics — that is, a time sequence in action is involved. The temporal characteristics of nerve-action patterns are practically infinite in number. Not only are they influenced by the number of nerve cells involved and by the self-limiting functions of each cell and synapse in the pattern, but by more general internal and external conditions. Thus, it is not surprising that we think, speak, or otherwise act at differing rates of speed, at different times. It is surprising, perhaps, that we demonstrate so many different forms of rhythmical activity.

1. *Self-Limiting Functions.* Basic to the problem of temporal pattern in nervous action are the self-limiting functions of nerve cells and synapses. We know that nerve cells conduct at different rates of speed, have different recovery rates, and demonstrate periods of relative refractoriness. We also know something of the matter of delay at synapses and appreciate the fact that the more synapses involved in a given stimulus-response circuit, the greater will be the conduction time. The problem of individual differences in rates of reaction should be investigated at these basic levels.

2. *Rhythmical Phenomena.* Rhythmic patterns of action in the nervous system have long been appreciated, but only recently are receiving thorough investigation. Of course, the rhythmical nature of the heart beat and the respiratory cycle are fairly well understood. But our knowledge of other rhythmical activity is not as satisfactory. What about tremors of the fingers or other parts of the body, — or what about nystagmus? What are their causes, their correlates, and their significance in human behavior? And, perhaps of even greater significance, what about the different rhythms of potentials from the brain that we have discussed in Chapters 19 and 23? All constitute fascinating problems in a partially explored field. You probably will hear much of them in the future.

3. *Compensatory Phenomena.* In this text you frequently have seen such terms as adaptation, adjustment, compensation, and homeostasis. All might be regarded as compensatory phenomena. The problem of sensory adaptation has already been mentioned. It also constitutes a form of compensatory phenomenon.

Many psychologists today are concerned with the problem of human adjustment, and the word is used in many different senses. Although we make many different adjustments almost entirely at the psychological level (e.g., we give up one goal in order to attain another), many of our adjustments in life are obtained at the physiological level. Furthermore, many of them assume a cyclic nature. For example, we engage in so much activity, become fatigued, and rest or sleep. We consume so much food and liquid, eliminate that portion of it that we do not utilize, and again become hungry or thirsty. Most closely related to rhythmical activity but still a compensatory phenomenon is our act of respiration. There are many other forms of compensatory behavior in which we engage but of which we are rarely conscious; such as the changes in our peripheral vasomotor system and sweating reactions to changes in external temperature, or the increased blood supply to muscles during periods of exercise. Many of these compensatory mechanisms are fairly well understood, of course, but the basic mechanism underlying others is understood in principle only. We have called it homeostasis, and have noted that it is related to compensatory changes in the two branches of the autonomic nervous system in particular. We have suggested that under conditions of stimulation the sympathetic branch usually is the one that is first brought into action, and that it is followed by secondary action of the parasympathetic branch, which then upon occasion may overcompensate. There is ample evidence that this pattern of events does occur and that it is based upon an underlying neural pattern, but we do not completely understand it. It is of great importance in the understanding of behavior and in particular in the understanding of some forms of psychosomatic disorders. It is important that we discover the underlying mechanism.

4. *Innate CNS Patterns in Man.* Man has long been concerned with the relative importance of inherited and acquired characteristics. Today, he is somewhat less concerned with this problem than 20 years ago, but in part this is due to the fact that he has been discouraged in his attempts to ferret out the effect of hereditary influences as divorced from learning. Although most of our human behavior patterns are learned, the potentialities for many of them are inherited. They must involve action patterns in the central nervous

system just as surely as do the sucking reflex and the breathing reflex shown by all new-born human infants. The problem of their description is difficult but will attract many investigators.

Problems of Biochemical Influences. Psychologists cannot escape a consideration of the influence of internal chemical conditions upon behavior. A paramount need in scientific investigation today is for more biochemists with psychological training and interests, or more psychologists with chemical training and interests, or, most desirably, both. Until a " meeting of the minds " in these two disciplines has been achieved, many important problems in physiological psychology will remain unsolved.

1. *On Nerve Cells and Nerve Nets.* Many drugs are known which will facilitate nervous action in general, inhibit it in general, or do either selectively for different nerve cells and combinations of cells. Doctors may give us adrenalin or atropine depending upon their desire to stimulate or to inhibit certain nervous patterns, or our drinking of common beverages may make us " jittery " or stuporous, as the case may be. When we know the manner in which these individual chemicals influence action in single nerve cells or in nerve nets, we shall know much more about the manner in which they influence patterns of nervous action and behavior.

2. *On Patterns of Nervous Action.* Certain chemicals seem to have differing effects on different parts of the nervous system. This must be due to the different chemical structure of the different parts, but the details are not known. The general anesthetics used in surgical operations afford one illustration of the matter we are discussing. They do not operate on all nerve cells in the body at the same time even though they presumably are being carried via the blood stream to all nerves at much the same time. Characteristically, their first action seems to be on higher nervous centers — those most recently added in evolutionary history. Gradually, with continued application, they exert action on lower and lower centers. There are many other illustrations. Anoxia affects the more rapidly metabolising structures first. Some drugs are known for their ability to produce dream states, others produce images of geometrical design, still others produce sensations of worms crawling over the body.

Here, too, lies the important field of relationships between endocrine secretions and behavior. What are the actual factors in pituitary secretion that influence migratory patterns of behavior? Or how may one explain the changed patterns in the mating behavior of some species that result from subtraction, and then addition, of gonadal hormones? Such relationships not only are significant, but they constitute fundamental problems in the understanding of behavior.

3. *On Muscle Action.* We know that muscular action is fundamentally due to alteration in chemical bonds. We have reason to believe, also, that the manner of stimulation of muscle by nerves is also partly dependent upon chemical media, and that certain chemicals (such as curare — the South American arrow poison) will block this action between nerve and muscle. Beyond this we know little, whether it be of individual action, or of individual differences in muscle actions between different people. Yet these are all important problems in any study of behavior, for the behavior of most organisms depends upon muscle action. Furthermore, muscle tension is a factor in personality and a form of motivation in human behavior. A closely related and important problem is that of fatigue. Muscles may become fatigued after constant action but we know that in many instances when we say " we are tired," we are actually referring to no real change in muscle functionability. In short, fatigue in human beings is not understood.

4. *On Organismic Behavior.* Although the problems mentioned in this chapter all relate to organismic behavior, it seems desirable to emphasize that certain internal biochemical states lead directly to certain patterns of behavior. As was pointed out in Chapter 20 these are motives, and their nature or their mode of operation is not always understood. For example, what is the nature of the food deficit drive and how does it lead to increased activity in organisms? And what about some of our learned addictions? Are internal chemical alterations associated with the motivation to smoke, to consume alcohol or coffee, or to take drugs? The answer, presumably, is yes; but we are not certain. These and many other problems in this field require further investigation. We cannot overemphasize the importance of the problems raised in this section on biochemical influences. Each cell must be regarded as a chemical factory, and almost every operation of it is chemical in nature. The study of cellular physiology is essentially chemical in nature and so, also, is the study of physiology in general. Physiological psychology, then, must await basic biochemical discoveries before it can solve many of its persisting problems.

Problems of Cortical Function and Behavior. The initial study of the effects on behavior of electrical stimulation of the cerebral cortex occurred less than a century ago. Since that time the experimental investigation of cortical function has occupied many investigators and it will continue to do so for years to come. Only a few of the significant research problems are mentioned here.

1. *Sensation and Perception.* Many of the important problems concerning sensation already have been solved, but at least two major areas of unknowns remain: (1) the problem of differential irritability of receptor cells, which has been treated above, and (2) the problem of quality in sensation. We ordinarily assume that the place in the cortex to which afferent impulses are projected determines the quality of sensation. Since the nerve impulses in all afferent fibers are believed to be the same, and since the receptor cell can be circumvented and different qualities of sensation can be produced by direct stimulation of the cerebral cortex, it would seem to follow that the projection area is the most important factor. However, we have to ask, how is it possible that if one place in the cortex is stimulated we have the sensation of odor while if another place is stimulated we have the sensation of vision? We need to discover the differences in cortical organization which underlie these sensory differences.

2. *Pleasantness and Unpleasantness.* Another perennial and important problem is the nature of pleasantness and unpleasantness in man. We know that both are of utmost importance in the determination of human behavior; but you will notice that we have made infrequent reference to either in this textbook. We have used " noxious " and defined a noxious stimulus in terms of overt, avertive behavior, but we do not have a comparable substitute for " pleasantness." Pleasantness and unpleasantness can be thought of as qualities of perception, but of a general nature. We have practically no information as to how such perceptions are mediated or of the manner in which they assist in determining other behavior. We do not understand completely the important sensation which we call pain, nor are we certain about the portion of the cortex which is responsible for it. We know that it determines much of our behavior, but we do not understand how.

3. *Learning and Memory.* Even less well understood are the all-important functions in man of learning and memory. We know that, for the most part, they are mediated by the cerebral cortex. But we do not know what changes in the nervous system underlie learning and we are not sure what regions of the cerebral cortex are primarily concerned with memory. Perhaps a better understanding of these problems will permit better control of learning.

4. *Imagination and Thought.* A recent novel was based on the interesting theme of attempting to define man. It concluded that man was to be distinguished from other animals in terms of his imagination and thought — particularly, his propensity to engage in self-contemplation and contemplation of the world about him. Many

other writers and philosophers have reached similar conclusions. Although comparative psychologists might differ and might say that subhuman animals do demonstrate both imagination and thought, they would agree that these abilities or activities are not shown in any other animal than man with the frequency and product demonstrated by him.

You probably know what you mean when you use either of these two terms, but ask the psychologist what he means and he will begin to equivocate. He not only has no adequate definition for either, he has no thorough understanding of the processes involved. He can say that there is good evidence that they are made possible by the frontal lobes of the cerebrum; but this conclusion is largely an inference. It is based upon the fact that it is in these frontal lobes that man's CNS differs most from other highly developed animals. Most of us would agree that creativeness and the capacity for reasoning are pre-eminent in intelligent behavior. But we have few if any adequate means for measuring them. True, we have many so-called intelligence tests. And, within their realm of application, they do a fairly adequate job. But the task of discrete measurement of the important functions of imagining and thinking is a primary one. When adequate measuring scales have been devised then we can begin to investigate the relationship of such abilities to cortical function.

5. *Consciousness, Hypnotic States, and Unconsciousness.* It is even more frustrating to consider the terms used to head this paragraph. You might be interested to know that the authors of this textbook spent their luncheon hours for a week attempting to arrive at a satisfactory definition of consciousness. The one finally published in the glossary satisfies none of us. Typical definitions employ the term "awareness," which is just as difficult to define. When we attend to a certain event in our environment we are aware of it but at the same time we are unaware of many other events. When we are distracted, we lose awareness of the original event and become aware of another. When we sleep, we become less aware of many stimuli but seldom, if ever, totally unresponsive. Presumably when we become unconscious, we become unaware — but still our central nervous system continues to react to many forms of stimulation as long as we continue to be living organisms. Under hypnosis, awareness can be made quite selective or, apparently, can be almost entirely eliminated. What kinds of processes are involved? At the moment no one knows. We do not even know the exact conditions which alter a waking state to a sleep state, or vice versa, although here, at least, a beginning has been made, as was seen in Chapter 23.

Presumably " consciousness " represents not a state but a continuum of excitability, from the most complete form of alertness in which an organism is irritable to practically any form of stimulation, to the most complete form of unawareness in which an organism is irritable to few stimuli. In the range then from the most extreme level of " consciousness " to the most extreme level of " unconsciousness " — short of death — would lie many different states. One portion of the range undoubtedly justifies the term " wakefulness "; another portion undoubtedly justifies the term " sleep "; but we probably must revise our thinking about the " either-or " character of these two states. Instead, we shall have to think in terms of degrees of wakefulness as determined by some objective criteria; or degrees of sleepfulness. Likewise we shall have to think in terms of degrees of consciousness, and degrees of unconsciousness. Presumably the states that may be induced through hypnosis may range throughout this continuum, but the validity of this statement can hardly be investigated until we have objective criteria for different levels. After that comes the very interesting problem of investigating the physiological correlates of hypnosis and the mechanism by which such states are achieved.

Physiological Factors in Abnormal Behavior. Abnormal behavior for a given species is defined, of course, as any behavior which deviates from the normal for that species. It is probable that any organism at some time during its life history will show one or another form of abnormal behavior. We, as individual human creatures, very often show abnormal behavior. We have a night when we do not sleep well, or we have a day when all we want to do is sleep. We eat too much, or do not eat enough, and our behavior suffers. Or occasionally we get queer " mental quirks." We begin to think that we are better than we normally are, or we begin to think that our abilities are deteriorating. Or even worse, we begin to think that people are plotting against us. Such behavior, if it becomes too extreme, or too consistent, becomes known as a " disorder." If we, ourselves, recognize it as abnormal we are apt to consult a physician, a psychiatrist, or a psychologist. If we do not regard it as abnormal, but others do, we are apt to be forced to consultation, or may even be committed to a psychopathic hospital. As was pointed out in Chapter 24, some forms of abnormal behavior are commonly recognized as attributable to physiological disorders, while others are regarded as psychogenic. It is our belief that all forms of abnormal behavior have their physiological correlates, and that in some cases, not now recognized, the physiological correlates are causal in nature. We would emphasize, then, the need to study physiological factors in psychopathology in general.

Fortunately, we are not alone in this belief. And here we see a curious phenomenon. Medical practitioners have, in the past, been loath to give due consideration to psychological factors involved in bodily disorders. Psychiatrists and clinical psychologists, on the other hand, have been loath to give due credit to physiological processes in connection with so-called " functional " disorders. Two different groups of consultants to the public have, then, been operating at extremes along what we regard as a continuum. Gradually these extremes are being extinguished. The average medical practitioner today is all too willing to admit the possibility of psychosomatic disorders. The average psychiatrist or clinical psychologist today is willing to admit that there are some organic factors in psychopathology which he does not understand. We see some real hope in the near future for physiological psychology to serve as a meeting ground for these two less extreme points of view. Both physiologists and psychologists, pure or applied, will contribute to this better understanding of abnormal behavior.

Since Chapters 22 and 24 have been devoted entirely to the consideration of mental disorder, only a few problems of general reference will be emphasized here. Among these the one creating the most research interest at the moment is " stress," and it is treated in some detail.

1. *Physiological Stress.* For a number of years psychologists have referred to " psychological stress." More often than not when they used such a term they implied that there was a conflict between two strong motives. Consider for a moment the child playing at the ocean side who has lost his rubber ball to the waves. He wants the ball, but it is floating several feet out in the water which he fears. His oscillatory behavior of approaching the ball and retreating from the waves is apt to be explained in terms of conflict between two motives. If both motives are strong, he is apt to end up in a dilemma which results in considerable physiological agitation, including the well-known childhood wail. But the latter form of behavior obviously involves physiological changes which, also, have had their precursors. In other words, it has come to be understood that we never have anything that can be called " psychological stress " without physiological involvement.

Over the years physiologists have gradually added meaning to the term " physiological involvement." First, it became realized that the autonomic nervous system was brought into a state of increased activity, particularly in its sympathetic branch. More recently, it has come to be known that still another system is activated. This system sometimes is referred to as the hypothalamic-hypophysial-adrenal-cortical system. We shall not go into its details here. Suffice it to say that with stimuli which strongly activate the sympathetic nervous system we typically have additional effects which involve hypothalamic stimulation of the pituitary gland (possibly via endocrine factors), the release of ACTH, the subsequent stimulation of the adrenal cortex, and the release of one or possibly more factors from this little understood endocrine gland. These adrenal-cortical hormones, in turn, set up adjustive processes in the body but, on occasion, may overdo their work. In other words, overcompensatory mechanisms may occur which, in turn, set up new processes of disorder within the organism. The whole response is called **stress.** Although the general pattern of this stress reaction has come to be known as "**the general adaptation syndrome**" (**GAS**), it is obvious that occasionally the adaptive mechanism gives way to one of overadaptation. Thus, a major need in today's psychophysiology is a detailed study of the manner in which the autonomic nervous system and the endocrine systems cooperate in both adapting an organism to a stressor, and in overcompensating. Stress may be produced either by psychological stressors, such as intolerable home or working conditions, or by the more direct physiological stressors such as extreme environmental temperatures or extreme and prolonged pain. A thorough consideration of the psychophysiology of stress may be expected to contribute much to our understanding of some forms of abnormal behavior.

2. *Psychosomatic Disorders.* We already have treated psychosomatic disorders at some length and shall not give much attention to them here. We wish to point out now only that overcompensatory mechanisms in connection with the stress response are believed by some workers to account for at least some of our psychosomatic disorders; in particular, the development of ulcers, the development of arthritis, and the development of certain skin diseases. Psychosomatic problems are of paramount importance in today's medical practice. But in this enthusiasm for the psychological aspect of medical disorders, we are apt to lose sight of the fact that basic physiology also is involved. Everyone does not develop an ulcer when stressed. Some get hives, some become asthmatic, some develop neurosis, and some just worry a little and get over it. What accounts for these individual differences? Certainly this problem is not solely psychological. It must be basically physiological. Here there are many questions but few answers.

3. *Hysteria.* One form of abnormal behavior which was among the first to be recognized and diagnosed is still among the least understood. It is known as hysteria. Hysteria is not as frequent in occurrence as some of our other neuroses but it is particularly interesting for two reasons: (1) it involves a physical symptom for which there is no known organic pathology, and (2) it seems so closely related to some of the phenomena we observe during hypnosis. We have many forms of hysteria — partial blindness, partial deafness, partial paralysis, and partial anesthesia — but typically they are partial. Rarely, if ever, do they involve complete loss of ability. Hysterical symptoms are known to develop during or immediately following times of great stress. That they do occur without the volition of the patient seems fairly clear. That they involve patterns of cortical activity also seems clear. And the same kind of a condition can be produced under hypnosis. The question is, what nerve patterns are involved and how are they controlled?

4. *Fatigue, Anxiety, and Other Neuroses.* We already have referred to fatigue (Chaps. 20, 21, and 23) and have suggested that it is related to emotional behavior and motivational conflict. Emotional behavior, however, is basically physiological, motivation is basically physiological, and strong conflict between motives produces physiological stress which in turn has other sequels. That " tired feeling " is seen, then, as a true psychosomatic disorder, whether or not it be classified as a neurosis. We need to have extensive biochemical studies of the chronically " tired " person. Without question the general feeling of tiredness has psychological components. Give such a tired person the opportunity of a diversion which really interests him and often he loses his fatigue. What physiological factors, however, are involved in this loss of fatigue? We can guess that some of them are related to the autonomic nervous system — we can even reverse his tired feeling by giving him a dose of adrenalin — but this does not solve the whole problem. Undoubtedly many other factors are involved, and until they are thoroughly studied from a psychophysiological point of view we shall continue to have millions of " tired " people among us.

The question of anxiety also has received previous attention (cf. Chaps. 20 and 21). You know that we consider anxiety to be a specific pattern of activation within the sympathetic branch of the autonomic nervous system. We have described it both as a pattern of emotional behavior and as a source of motivation. If these analyses are correct, it should be possible, theoretically, to control anxiety neuroses. The basic element would seem to be a factor which will reduce the activity of the SNS. The theoretical ground work is laid; the essential experiments are now being conducted. It is too early to speak of the results.

5. *The Psychoses.* The psychoses, the true insanities, present even more difficulties of understanding than do the neuroses. Some of them are obviously physiological in nature (see Chap. 24). But beyond this we know little. What accounts for the systematized delusions that are shown occasionally in the disorder that is diagnosed as true paranoia? What accounts for the oscillatory swings from the depths of depression to the height of elation, as frequently seen in the disorder known as manic-depressive psychosis? And, perhaps even more significant in terms of the frequency of the disorder, what accounts for the regressive behavior demonstrated by the schizophrenic?

Mood shifts are common in many of us. We oscillate between mild degrees of depression and mild degrees of elation. On some days we function better than we do on others. The factors underlying these differences in behavior have never been adequately studied. When they are we may have some additional insight into the mechanisms of manic-depressive psychosis. Without question they are physiological in nature. Though they may have their psychological setting, and it may be that which actually induces them, they will not be induced in everyone. Any understanding of manic-depressive psychosis, therefore, must go back not only to a study of moods and temperament but to a study of individual differences in patterns of activation in the nervous system. This is, basically, a problem in biochemistry and physiology. Psychology can do little with it until the underlying elements are better understood.

Schizophrenia is even less well understood. It probably is not a good term; it covers too broad an area and includes such different characteristics as regression in behavior, retreat from society, depression, or marked agitation — particularly in the specific disorder known as paranoid schizophrenia, with delusions of persecution or of grandeur. In the last two or three decades, such psychotic states often have been treated by some form of shock to the central nervous system. These treatments have taken the form of insulin shock, metrazol shock, or electrical shock. In some instances the use of one of these forms of treatment seems to have rehabilitated some patients who otherwise might have spent the rest of their lives in psychopathic hospitals. Presumably all have similar effects upon the nervous system; but it is to be regretted that we do not know more about the manner in which any one of these convulsive forms of therapy exerts its effect. Electrical shock is used more today, not because it is known to be more generally effective, but because it is easier to apply and easier to control. Such forms of therapy provide stimulation to the nervous system in general; and in those individuals who demonstrate lethargic reactions to stimuli, these forms of shock therapy seem to be most effective. We infer, therefore, that shock or convulsive therapy does something to revivify the activities of the nervous system. We need to know what and how.

Concluding Comment. The problems we have outlined above are only a few among the many facing physiological psychology and its related disciplines. In the near future many of them will be solved, for at no other period in history has there been so much support for basic research, nor so much general interest in scientific productivity. We hope your acquaintance with this book will better enable you to appreciate the new information in this ever-changing field.

Appendix

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Glossary

Aberration (optical): Unequal refraction of light; light coming from a single source is not brought to a single focus.

Ablation: The removal of tissue.

Absolute refractory period: The very brief period after excitation in a neuron during which no response can be evoked.

Accommodation (visual): Adjustment of the lens of the eye for sharp vision at various distances; it is accomplished by changes in tension of the ciliary muscles.

Acetylcholine (ACH): A chemical substance which is present in much nervous tissue, and is known to be liberated by preganglionic ANS fibers, postganglionic PNS fibers, and those efferent fibers innervating striated muscle tissue.

ACH: (*See* acetylcholine.)

Achromatic color: A visual sensation which has no hue; the black-gray-white series of colors.

Acromegaly: Abnormal growth after puberty; excessive lateral growth in a fixed skeleton.

ACTH: (*See* Adrenocorticotrophin.)

Actomyosin: The complex chemical substance in a myofibril, responsible for muscle contraction.

Acuity (sensory): Fineness of sensory discrimination.

Adaptation: A progressive change in sensory response following continued stimulation.

Adaptation, attentional: Decrease in response to a stimulus due to attending to something else.

Adenohypophysis: (*See* Pituitary, anterior.)

Adequate stimulus: The normal stimulus for a given sense organ.

Adrenal cortex: The outer layers of the adrenal glands; secretes a number of hormones, collectively called cortin or cortical steroids.

Adrenal glands: A pair of endocrine glands resting on the kidneys (*see* Adrenal cortex and Adrenal medulla).

Adrenalin: A hormone of the adrenal medulla; usually synonymous with epinephrine.

Adrenal medulla: The centers of the adrenal glands; a separate endocrine gland that secretes epinephrine and norepinephrine.

Adrenergic: An adjective applied to substances which produce adrenalin-like effects, or to organs that respond to adrenalin.

Adrenocorticotrophin: The hormone of the anterior pituitary which stimulates the adrenal cortex to release some cortical steroids. Also called ACTH.

Afferent: Pertaining to the conduction of nerve impulses from the periphery of the organism toward the CNS and, within the CNS, from caudal elements toward the cerebral cortex.

After-sensation, negative: After-image in which the qualities are the complements of those induced by the stimulus.

After-sensation, positive: After-image in which the qualities are the same as those induced by the stimulus.

Ageusia: Inability to taste a particular quality or substance, taste-blindness.

Agnosia: Inability to recognize objects; may be primarily in one sense, e.g., visual agnosia.

All-or-nothing law: A generalization stating that if a neuron responds at all it will respond with the maximum energy possible at the given time. The law is sometimes applied also to single muscle cells.

Allocortex: Older portion of the cerebral cortex, closely associated with olfaction.

Alpha blocking: Disappearance of the alpha rhythms as the result of stimulation.

Alpha rhythms: In the adult human, EEG rhythms of about 8 to 12 per second.

Amino acid: A constituent of protein containing carbon, oxygen, hydrogen, and nitrogen; there are several different amino acids.

Ampulla: Enlargement of a semicircular canal which contains the receptor cells.

Anabolism: That aspect of metabolism which involves the building-up or restoration of protoplasm.

Analgesia: Absence of the sense of pain.

Analgesics (*sing.* analgesic): Drugs which reduce the sensitivity to pain.

Androgen: A collective term for the male gonadal hormones (*see* Testosterone).

Anion: The negative ion, the one which moves toward the positive pole or anode.

Annulo spiral ending: A receptor of the muscle spindle which probably does not give rise to impulses reaching awareness.

Anosmia: Inability to smell, either totally or a particular substance or group of substances.

Anoxia: General oxygen lack, from one of several causes.

ANS: (*See* Autonomic nervous system.)

Antidiuretic hormone: A hormone of the posterior pituitary which inhibits the production of urine.

Antidromic: An adjective applied to a nerve impulse which originates in an axon process and is conducted toward the cell body of a neuron or reaches another axon termination.

Antipyretic: A drug which lowers the body temperature or reduces fever.

Aphasia: Difficulty in recognizing or producing language.

Aqueous humor: A fluid which fills the space between the cornea of the eye and the lens.

Arachnoid: Delicate, middle meningeal layer.

Association cells: Neurons other than afferent or efferent neurons.

Auditory ossicles: The three small bones bridging the middle ear from tympanum to oval window.

Aural harmonics: Sounds generated within the ear itself.

Autonomic nervous system (ANS): That portion of the nervous system, both central and peripheral, which sends efferent fibers to smooth muscles and glands.

Axon: The neuronal process or fiber which ordinarily conducts impulses away from the cell body, toward other neurons or effector cells.

Basal ganglia: The corpora striata.

Basal metabolism test: A measure of the rate of oxygen consumption in a resting, fasting state; regarded as a measure of function of the thyroid gland.

Beats: Rhythmic waxing and waning generated when two tones of slightly different frequencies and about the same intensity are sounded together.

Behavior: The activity of organisms.

Bel: Unit of sound intensity. Now defined as the $\log_{10}$ of the ratio between the given sound intensity and .0002 dynes per cm^2.

Bends: Pain and tissue damage from rapid reduction in environmental pressure.

Beta rhythms: Rhythms in the EEG of a frequency of about 25 per second.

Bimodal distribution: A distribution of scores showing two peaks or modes.

Binaural: Refers to both ears, as in binaural stimulation (stimulation of both ears at the same time).

Bipolar cells: Cells in the retina which connect the visual receptors with the ganglion cells; also, in general usage, any neuron with one axon and one dendrite.

Blood-brain barrier: All the collective structures through which a substance in the blood must pass to reach the interior of a cerebral neuron.

Bodily image: The person's perception of himself.

Boredom: Psychological fatigue in the absence of significant previous muscular activity.

Brain: The central nervous system above the spinal cord.

Brain stem: The brain exclusive of the cerebral and cerebellar hemispheres.

Brightness: One dimension of visual sensation (or color); the light-dark aspect; the similarity to white.

Bronchia: The passages in the lungs.

Calorie: The large calorie is the amount of heat necessary to raise 1 kilogram of water from 0° to 1° C.

Cardiac muscle: The specialized muscle tissue found only in the hearts of mammalian organisms and others with true circulatory systems.

Catabolism: That aspect of metabolism which involves the expenditure of energy with a breakdown of protoplasm.

Cation: The positive ion, the one which moves toward the negative pole or cathode.

Caudal: Pertaining to the tail region of an organism.

Causalgia: A severe, persistent, burning pain, with disturbance of temperature and abnormal skin appearance in the affected area.

Cell (in Biology): The unit structure in organisms, which may be single-celled (Protozoa) or multicelled (Metazoa).

Cell, receptor: (*See* Receptor.)

Cell body: As used in this text, that portion of a neuron which contains the nucleus and associated structures, and from which extend the dendrites and axon.

Central nervous system (CNS): That portion of the nervous system which lies within the skull and spinal column; the brain and spinal cord.

Cerebellum: A central nervous system structure lying dorsal to the pons and beneath the cerebrum.

Cerebral cortex: The surface layer of gray matter of the cerebral hemispheres.

Cerebral hemispheres: The lateral halves of the cerebrum.

Cerebrum: The uppermost or most anterior part of the central nervous systems; those parts of the brain above the diencephalon.

CFF: (*See* Critical flicker frequency.)

Chemical integration (of behavior): The ordering of behavior via hormones and other chemical substances within the body.

Cholinergic: An adjective applied to substances which produce acetylcholine-like effects, or to organs that respond to acetylcholine.

Cholinesterase: Any of several enzymes which cause the breakdown of acetylcholine.

Choreoathetosis: Disorder of movement characterized by involuntary circular or writhing motions.

Chorionic hormone: A hormone secreted by placental tissues in pregnancy which serves to maintain the state of pregnancy.

Choroid: The middle coat of the eye, a layer containing blood vessels, and becoming the iris at the front of the eyeball.

Choroid plexus: Specialized vascular structure in a ventricle of the brain which secretes cerebrospinal fluid.

Ciliary muscles: Muscles in the eye which change the shape of the lens, permitting variable focus.

Cilium (pl. cilia): A short, hair-like projection on a cell.

Climacteric: The cessation of reproductive processes; in the female this is often called the menopause.

Clonus: A series of rapid involuntary contractions and relaxations in a muscle.

CNS: (*See* Central nervous system.)

Cochlea: Auditory portion of inner ear.

Cochlear potentials: Changes in electrical potential which may be recorded from the cochlea.

Colliculi (superior and inferior): Two pairs of small protuberances on the dorsal side of the midbrain, of which the uppermost are called superior and the lower inferior.

Color: The general term for visual sensation. It is usually used for hue in popular parlance, but hue is only one dimension of color.

Coma: State of complete unresponsiveness from which the organism cannot be aroused.

Combination tones: Tones generated within the auditory mechanism, by two primary tones, which are heard as tones with frequencies corresponding to the sums and differences of the two primary tones and their harmonics.

Common chemical sense: The sense in the mucous membranes which responds to certain chemical agents, probably identical with pain.

Complementary (colors): Colors which will give gray when their stimuli are mixed.

Conditioned response: A response which comes to be elicited by an originally neutral stimulus.

Conditioned stimulus: A stimulus, originally ineffective, which by association with a US becomes effective in eliciting behavior.

Conductivity: The ability to propagate a nerve impulse or other physical change from one point to another; an ability demonstrated by nerve cells and other less specialized protoplasm.

Cone: One of two types of visual receptors; cones function in high illumination and subserve color vision.

Connector: In the primitive nervous system, a neuron between receptor and effector.

Consciousness: A term used to describe an individual organism while it is perceiving, remembering, thinking, imagining.

Contractility: The ability to shorten, or to exert pull if attached; an ability demonstrated by muscle cells and other less specialized protoplasm.

Contraction, isometric: Contraction of a muscle without shortening.

Contraction, isotonic: A contraction in which a muscle shortens.

Contraction, phasic: Any tetanic contraction in muscle which is not maintained.

Contraction, tetanic: (*See* Tetanus.)

Contraction, tonic: Any sustained tetanic muscular contraction.

Convergence: The turning of the eyes inward so that near objects may be fixated.

Cornea: The transparent part of the outer coat of the eyeball, which covers the front of the eye and admits and refracts light.

Corpus luteum: The yellow, or luteal, body which develops in the follicle after ovulation and secretes progesterone.

Corpus striatum (*pl.* corpora striata): The basal ganglia of the cerebral hemispheres; large nuclei concerned with some motor functions.

Cortex: A surface layer of cell bodies on some organ, cf., cerebral cortex, cerebellar cortex, adrenal cortex.

Cortical steroids: The hormones of the adrenal cortex, sometimes called cortin.

Cortical suppressor area: Cortical area stimulation of which leads to reduction of activity in another cortical area or areas.

Cortin: (*See* Cortical steroids.)

Covert: An adjective applied to internal activities of organisms that ordinarily can be observed only with the aid of special instruments.

CR: (*See* Conditioned response.)

Cranial nerve: Any of a dozen pairs of nerves which enter or leave the brain rather than the spinal cord.

Cranio-sacral system: The anatomical term for that portion of the ANS which involves nerve fibers coming from the brain stem and sacral portions of the spinal cord (*see* Parasympathetic nervous system).

Cretinism: A state in which there is deficient thyroid secretion at birth or during the early years of life.

Crista: The mass of receptor cells in the ampulla of a semicircular canal.

Critical flicker-frequency (CFF): The frequency of interruption of a light at which the separate light-dark phases are no longer seen and only a homogeneous light appears.

CS: (*See* Conditioned stimulus.)

Cupula: Gelatinous mass associated with the crista in ampulla of semicircular canals.

Curare: A drug which blocks the neuromyal junction and thus paralyzes striated muscles.

Cutaneous: Of the skin.

Cycle: One complete revolution or one complete oscillation of a periodic event.

Cytoarchitectonics: Study of the distribution of cells and cell types in an organ.

Cytology: The study of cells.

Cytoplasm: The material of living cells that is contained within the cell membrane, exclusive of the nucleus.

db: Abbreviation for decibel.

Decerebrate rigidity: Extreme extensor tone shown after section of the brain stem above the vestibular nuclei.

Decibel: One tenth of a bel.

Delta rhythms: Rather slow rhythms

in the EEG, characteristic of light sleep.

Dendrite (typically *pl.*): One or more processes or fibers projecting from the cell body of a neuron which ordinarily are excited across a synapse and conducted toward the cell body.

Dermatome: The skin area supplied with afferent neurons by a given posterior root.

Deuteranopia: A form of red-green color blindness in which the visibility curve is normal but reds and greens are confused.

Diabetes mellitus: A physical disorder caused by insulin deficiency.

Diabetic coma: Unconsciousness caused by insulin deficiency.

Diathesis: Predisposition toward disorder in a given organ, inherited or acquired.

Dichromatism: Partial color blindness; either red-green blindness or blue-yellow blindness; two primary colors are required to match all colors.

Diencephalon: The uppermost portion of the brain-stem, consisting in large part of the thalamus and hypothalamus.

Diplacusis: Perception of different pitches through the two ears with the same stimulus.

DL: From the German Differenz Limen (*see* Difference threshold).

Dorsal: Pertaining to the back of an organism.

Dorsal root: Bundle of afferent nerve fibers entering the spinal cord; joins with a ventral root (q.v.) to form a spinal nerve.

Dorsal root ganglion: The enlargement on a dorsal root caused by a concentration of cell bodies of bipolar afferent neurons.

Drive: An unlearned motive.

Dromic: An adjective applied to a nerve impulse which is conducted from dendrites and cell body toward axon terminations.

Dura mater: Tough, outer meningeal layer.

Ear, inner: Portion of ear lying in the skull, and consisting of an auditory portion (the cochlea) and a proprioceptive portion (the vestibular organs).

Ear, middle: Air-filled chamber between eardrum and inner ear.

Ear, outer: The portion of the ear peripheral to the ear drum.

Efferent: Pertaining to the conduction of nerve impulses from the cerebral cortex to lower centers but, more specifically, from the spinal cord and brain stem to muscles and glands.

Effector: A cell, tissue, or organ that is specialized for some form of activity other than irritability or conductivity; i.e., usually contraction or secretion.

Electrical field: As used here, refers to the changes in electrical potential surrounding an electrically charged portion of a cell membrane.

Electroencephalogram (EEG): A record of the electrical activity of the brain recorded from outside the skull; brain waves.

Electrolyte (in biology): A chemical substance within a living cell which can form ions carrying either positive or negative electrical charges.

Electromagnetic spectrum: The various wavelengths of radiation, varying, so far as is known at present, from 100^{-10} cm. (gamma rays) to 3×10^6 cm. (radio waves).

Electromyogram: A record of action potentials in muscle. Sometimes called EMG.

Elimination (in biology): The processes by means of which organisms rid themselves of the waste products of metabolism and of other unusable matter.

EMG: (*See* Electromyogram.)

Emotion: Internal or visceral activity (*see* Emotional behavior).

Emotional behavior: Behavior that is activated by the ANS.

Emotional expression: Overt muscular activity elicited either by emotion or by emotional ideation.

Emotional feeling: The perception of emotion.

Emotional ideation: An intellectual response which originally accompanies emotion, but which, through learning, may subsequently occur in the absence of emotion.

Encephalization: Tendency, during the course of evolution, for the more anterior parts of the CNS to show the greatest development and to assume dominance over lower structures.

Endocrine gland: An organ of the

body which produces and secretes into the blood stream a chemical substance which affects another part of the body.

Endocrinology: The science of endocrine gland function.

Endolymph: Liquid filling the cochlea and vestibular organs.

Epilepsy: A group of convulsive disorders, often associated with clouding or loss of consciousness and changes in EEG pattern.

Epinephrine: A hormone of the adrenal medulla; usually synonymous with adrenalin.

Epithelium: Tissue which forms the skin and lines the hollow organs and passages of the body.

Equal loudness contours: Lines drawn on a chart which show the amount of stimulus energy necessary at each frequency in order that sounds at the different frequencies shall be equally loud.

Equilibratory behavior: Behavior that appears to be directed toward the reestablishment of a state of equilibrium in an organism.

Equilibrium, physiological: (*See* Homeostasis.)

Erogenous zones: Skin areas in which stimulation usually leads to sexual excitement (especially genitalia, nipples, and lips).

Estrogen: A collective term for the primary female gonadal hormones.

Estrus (sometimes spelled oestrous)**:** A period at which nonprimate female mammals are easily excited by, and receptive to, males of their species; often called heat; usually associated with ovulation.

Eustachian tube: Tube connecting the middle ear with the throat. It serves to equalize the air pressure on the two sides of the eardrum.

Excitability: Irritability; the property of responding to one or more kinds of energy change.

Excitation: Refers to the process of arousing or irritating a cell to heightened activity; the opposite of inhibition.

Extensor: A muscle which extends a part of the body.

Extensor thrust: Extension of a limb as the reflex result of stimulation of the proprioceptors or pressure receptors of the toe pads.

External auditory meatus: Canal from outside of head to the eardrum.

Extrapyramidal: Adjective referring to motor tracts or structures not in the pyramidal system, q.v.

Fatigue, physiological: Reduction in the capacity of an organism to do work as the result of previous activity. Impairment, q.v.

Fatigue, psychological: A feeling or perception of tiredness that usually increases with work output and with time elapsed after rest and sleep.

Final common path: The motor neuron connected to a group of muscle fibers.

Flagellum (*pl.* flagella)**:** A long hairlike projection from a cell, which often serves as a means of locomotion in protozoa.

Flexion reflex: Reflex bending of a limb when receptors in it are stimulated.

Flexor: A muscle which flexes a joint or pulls a part of the body inward.

Flicker: In vision, the fluctuating visual impression resulting from the slow alternation of stimulation, usually light versus dark phases.

Flight of colors: A type of aftersensation produced in vision by an intense stimulus; there is a changing sequence of colors.

Flower-spray ending: Receptor of muscle spindle whose activity may result in awareness.

Follicle (ovarian): The tissue growth within the ovary which encloses the developing female germ cell and, later, the luteal body.

Follicle-stimulating hormone: The hormone of the anterior pituitary which is primarily responsible for the growth of the germ cells in both sexes and, indirectly, for the development of sexual characteristics; also called FSH.

Fovea: The central depression in the retina, where light rays are focused when one fixates an object. Region of clearest daylight vision.

Frequency: Number per unit time, e.g., number of cycles per second of a sound wave.

FSH: (*See* Follicle-stimulating hormone.)

Functional integrity: As used in this text, a state of an organism in which no function is impaired.

Functional psychoses: Those psychoses for which no organic basis has been demonstrated.

Fundamental: Lowest or basic frequency of a sound.

Galvanic skin response: A decrease in the electrical resistance of the skin, usually attributed to the action of the sweat glands.

Ganglion: A term typically used to describe groups of cell bodies lying outside the CNS.

Ganglion cells (retina): The third-order neurons in the visual system. They are connected to the bipolar cells, and their axons form the optic nerve.

GAS: (*See* General adaptation syndrome.)

Gastrointestinal tract: The digestive tract, from mouth to anus.

General adaptation syndrome: A term sometimes used to denote the stress response (*see* Stress); including phases of shock, counter-shock, specific recovery, and sometimes exhaustion.

General paresis: A deteriorated state due to an advanced stage of syphilis of the brain.

Gland: An organ of the body specialized for secretion.

Glial cell: (*See* Neuroglia.)

Glucose: A simple sugar; a form of carbohydrate released by the liver and readily utilizable by other cells of the body.

Glycogen: The form of carbohydrate which is stored in the body, principally in the liver, for later use.

Goal: Object, condition, or state toward which an organism is motivated.

Gonad: A generic term referring to the sex glands of either sex (*see* Ovary and Testis).

Gonadotrophic hormones: Those hormones of the anterior pituitary which stimulate the gonads to produce and release germ cells and hormones; specifically the hormones FSH and LH.

Grand mal: A severe form of epilepsy, involving convulsion, in which the patient loses consciousness for an appreciable period and may be injured; it is followed by a deep sleep.

Gray matter: A term used to describe those portions of the nervous sytem (the CNS in particular) that are gray in appearance; cell bodies and other unmyelinated portions of the nervous system.

Growth: A change in an organism correlated largely with the passage of time; not the result of experience or learning; maturation.

Growth hormone: A hormone of the anterior pituitary which facilitates growth of tissues; sometimes called somatotrophic hormone, or STH.

GSR: (*See* Galvanic skin response.)

Gustation: The sense of taste.

Gyrus (*pl.* gyri): Term applied to the ridges on the surface of the cerebral cortex.

Harmonics: Overtones.

Homeostasis: The observed tendency of organisms to maintain internal equilibrium.

Homologous series: A series of chemical compounds which are closely related, but differ systematically (e.g., the primary alcohols).

Hopping response: Alternate flexing and extending of leg when foot is dragged across a surface while animal is suspended.

Hormone: A chemical substance produced in an endocrine gland.

Hue: One dimension of visual sensation (or color). The redness, blueness, greenness, etc.

Hydrocephaly: A condition resulting from excessive cerebrospinal fluid in the ventricles.

Hyperalgesia: Increased sensitivity to pain.

Hyperhydrosis: Excessive perspiration, usually from the hands, feet, and armpits.

Hyperthyroidism: Hyperactivity of the thyroid gland.

Hypoglycemia: Below-normal blood sugar level.

Hypomania: A state of abnormal excitement, not as profound as true mania.

Hypophysis: (*See* Pituitary, anterior and posterior.)

Hypothalamus: The most ventral portion of the diencephalon.

Hypothyroidism: Hypoactivity of the

thyroid gland (*See* Cretinism and Myxedema).

Hysterical: Applied to certain organic symptoms when they are believed to have a psychological cause.

Hysteria: A form of neurosis in which some form of bodily disorder becomes the major symptom.

Impairment: Reduced function, sometimes used to mean physiological fatigue.

Incentive: An external goal-object toward which an organism is motivated.

Incus: Intermediate auditory ossicle, the anvil.

Inhibition: In general, the opposite of excitation. The term has many uses but here it usually means a reduction in or prevention of response as a result of stimulation.

Innervated: An adjective used to denote that a given effector cell, tissue, or organ is supplied with one or more neurons which may excite or inhibit it.

Innervation ratio: The number of nerve fibers innervating a given muscle, divided by the number of muscle fibers involved.

Inorganic compounds: Chemical compounds not containing carbon.

Instinctive behavior: A pattern of activity that is common to a given species and that occurs without opportunity for learning.

Insulin: The hormone of the islet cells of the pancreas.

Insulin shock: Unconsciousness caused by insulin excess.

Introspective technique: Psychological method in which the observer (subject) describes his awareness to the experimenter.

Iodopsin: The photosensitive pigment in the cones of the retina.

Ion: An electrified particle of an electrolyte which may carry either a positive, or a negative, charge; positively charged ions are called *cations*, negative ions are called *anions*.

Iris: The structure surrounding the pupil of the eye, which contains two sets of muscles controlling the diameter of the pupil; it contains pigment which gives color to one's eyes.

Irritability: The property of responding to one or more kinds of energy change.

Islet cells: Inner cells of the pancreas; an endocrine gland which secretes insulin; sometimes called Islands of Langerhans.

Isomorphism: Relation of two regions having the same form.

JND: Just-noticeable-difference, or difference limen (DL).

Kappa rhythms: Rhythms in the EEG of about 10 per second detectable of the temporal lobes.

Kinesiology: The science of movement. Usually refers to the study of the mechanical (muscle and joint) aspects of movement.

Kinesthesis: Muscle, tendon, and joint sensitivities.

Knee jerk: The patellar reflex, q.v.

Lactogenic hormone: A hormone of the anterior pituitary which facilitates the production of milk by the mammary glands; sometimes called prolactin.

Learning: Any more than transient modification of behavior which presumably results from past experience and not from known organic change.

Lens: A transparent structure within the eye which focuses light rays. It is adjustable so that rays coming from both near and far objects can be focused upon the retina.

Lesion: Region of destruction or abnormal changes in structure.

LH: (*See* Luteinizing hormone.)

Limen: (*See* Threshold.)

Lobotomy, prefrontal: Surgical separation of the prefrontal lobes from influence on or by the thalamus.

Local excitatory process: The wave of negative potential recordable from a neuron before a spike potential occurs, or when excitation is just subliminal. In the latter case, it is localized in the stimulated region of the nerve fiber.

Loudness: Psychological dimension of sound most closely related to sound wave intensity.

Luteal body: (*See* Corpus luteum.)

Luteinizing hormone: That hormone of the anterior pituitary which stimulates the secretion of progesterone from the luteal bodies of the female ovaries, and testosterone from the testes; also called LH.

Maculae (*sing.* macula): Receptor regions in saccule and utricle.

Malleus: The auditory ossicle nearest the tympanum. The hammer.

Manic-depressive psychosis: A profound behavioral disturbance, which may be characterized by an excited phase, or a depressed phase, or alternations of excitement and depression.

Masking (auditory): The raising of the threshold for one tone by the introduction of another.

Medulla (oblongata): That portion of the brain stem lying next above the spinal cord (also called the myelencephalon).

Meissner's corpuscle: One of the encapsulated end organs in the skin.

Melancholia: Extreme depression.

Melancholia, involutional: Depression which occurs in middle life, sometimes thought to be due to the hormone changes which accompany the cessation of reproductive processes.

Membrane, basilar: A thick, fibrous membrane running the length of the cochlea and bearing on it the organ of Corti.

Membrane, Reissner's: A thin membrane dividing two of the canals within the cochlea.

Membrane, tectorial: Small membrane in the cochlea adherent to the ends of hair cells of the organ of Corti.

Meninx (*pl.* meninges): One of the three membranes surrounding the CNS (*see* Arachnoid, Dura mater, and Pia mater).

Metabolism: The life process of cells and organisms, including the building up of protoplasm (anabolism) and its destruction (catabolism).

Metazoa: Multicelled organisms.

Microphonics, cochlear: Electrical responses generated in the organ of Corti of the inner ear.

Midbrain: That portion of the central nervous system lying between the pons and the diencephalon (sometimes known as the mesencephalon).

Millilambert: One-thousandth of a lambert, which is the brightness of a surface which is reflecting one lumen per square centimeter. (A lumen is the amount of light emitted in a unit solid angle by a point source of one international candle.)

Millimicron: A millionth of a millimeter; ordinarily abbreviated mμ.

Modalities (*sing.* modality): Refers, in this case, to a sense department; i.e., a sense, such as touch, vision, etc.

Molar concentration: Gram molecules per liter of solution (thus, a substance with molecular weight 30 would make a 1-molar solution in water if there were 30 grams of the substance in each liter of the solution).

Monaural: Refers to one ear, as in stimulation of one ear at a time.

Monochromatism: Total color blindness; lights of all wavelengths are seen as grays.

Motivation: A term employed to account for other than reflexive behavior initiated or controlled by conditions within an organism.

Motive: An internal organismic state which initiates or otherwise determines behavior.

Motor end-plate: A specialized receptive area of the sarcolemma; the muscular portion of the neuromyal junction.

Motor nuclei: Groups of cell bodies of efferent nerve fibers, within the CNS.

Motor unit: A single efferent nerve fiber and its associated muscle fibers.

Mountain sickness: Illness from high altitude, characterized by restlessness and poor sleep, irritability, and feeling of discomfort.

Movement, ballistic: In the pure case, a movement initiated by a muscular contraction but continuing because of momentum.

Movement, controlled: A movement during which both agonist and antagonist maintain tension.

Movement, fixed: Sustaining of a fixed position by means of muscular contraction.

Multipolar cell: A neuron which has an axon and two or more dendrites.

Muscle: A tissue specialized for contraction.

Muscle bundle: A group of muscle cells surrounded by sheaths of connective tissue.

Muscle cell: The structural unit of muscle tissue.

Muscle spindle: Receptor structure in muscle. *See* Annulo-spiral and Flower-spray endings.

Myelin sheath: The white fatty sheath that surrounds the axons of many neurons and provides the white color to many portions of the nervous system.

Myofibril: The thread-like contractile element in a muscle cell.

Myxedema: A state of deficient thyroid secretion.

Narcosis: Stuporous condition resulting from drug action; the process leading to such condition.

Narcotic: A drug which produces stupor.

Nasal mucosa: The membranous linings of the nasal passages.

Need: Any factor necessary for the achievement of a stated or implied goal.

Need state: A specific state within an organism or a machine, related to purpose, which may elicit behavior appropriate to the need.

Negative after-potential: A brief secondary wave of negative voltage that may occur in some neurons shortly after the spike potential.

Neocortex: The more recently evolved portion of the cerebral cortex.

Nerve: A bundle of nerve trunks, some of which may be afferent and some efferent.

Nerve cell: The structural unit of the nervous system; consisting of a cell body, an axon, and often dendrites (syn. neuron).

Nerve fiber: A process of a neuron that extends from the cell body (typically, an axon).

Nerve impulse: The electro-chemical change which is propagated along a neuron.

Nerve net: A group of neurons which function together (1) as a simple nervous system in primitive organisms, (2) as a closed unit in a complex organism.

Nerve trunk: A bundle of nerve fibers (typically, axons).

Neural fold: Ridged groove along the dorsal side of the vertebrate embryo which develops into nervous tissue.

Neural tube: Stage following neural fold in the embryonic development of the nervous system. The fold has closed to form a hollow tube.

Neurilemma: A thin sheath that covers neurons outside the CNS, except at their peripheral terminations.

Neuroblast: Embryonic neuron.

Neuroglia: Supporting non-neural cells of the CNS.

Neurohypophysis: (*See* Pituitary, posterior.)

Neuromuscular junction: (*See* Neuromyal junction.)

Neuromyal junction: Point of functional contact between neuron and muscle cells. Sometimes called myoneural junction and neuromuscular junction.

Neuron: The unit structure of the nervous system; a nerve cell, including a cell body and its processes or fibers.

Neuropil: Area in CNS of mossy appearing intertwining dendrites and axons.

Neurosis: A behavioral disturbance characterized by personality disorders and sometimes by disordered function of bodily organs.

Noise: Aperiodic sound.

Nor-epinephrine: A hormone of the adrenal medulla; also nor-adrenalin.

Normal distribution: A statistical term signifying that the frequencies of observations of a given function, when plotted, assume a normal or bell-shaped curve.

Noxious: An adjective applied to any stimulus that is followed by avoidance behavior.

Nuclear delay: The time required for the first postsynaptic impulses to appear from a nucleus when the nucleus is stimulated by an asynchronous barrage of presynaptic impulses.

Nucleus (of a cell)**:** Structure essential to the life of the cell; contains the chemicals determining heredity.

Nucleus (nervous system)**:** A group of cell bodies within the CNS.

Nucleus, cochlear: Nucleus in the medulla which serves audition.

Nucleus, vestibular: Nucleus of the medulla and pons related to the vestibular sense.

Nystagmus, physiological: Minute involuntary tremors, drifts, and jerks of the eyes which occur even during fixation.

Occlusion: A phenomenon in reflex activity due to overlapping connections of afferent neurons so that stimulating two afferent nerves simultaneously leads to a reflex response less than the sum of the responses obtained individually.

Oculogyral illusion: Illusory movement of a faint light when the subject is rotated, even though the light is rotating with the subject.

Olfaction: The sense of smell.

Olfactory epithelium: Pigmented layer in the nose containing the receptor cells for olfaction.

Optic chiasm: The point of crossing of the optic nerve fibers from the nasal or inside halves of the two retinae.

Optic disc: The blind spot, or point of exit of the optic nerve. It contains no visual receptors.

Organ: A bodily structure composed of one or more kinds of tissue and usually specialized in function.

Organ of Corti: Structure in inner ear bearing the hair cells, the receptors for audition.

Organic compounds: Chemical compounds containing carbon.

Organism: A single-celled or multicelled animal or plant whose structure permits life.

Oval window: Point of entry of sound vibrations into the inner ear; it consists of a membrane-covered opening to which the stapes is adherent.

Ovary: The female gonad; develops female germ cells and hormones (*see* Estrogen and Progesterone).

Overt: An adjective applied to behavior that can be observed without the aid of special instruments.

Overtones: Frequencies which are some whole-number multiple of the fundamental.

Ovulation: The time at which an ovum is released from the follicle and ovary.

Ovum (*pl.* ova): A female germ cell.

Oxygen debt: Said to occur when a process leads to consumption of extra oxygen after the process has terminated; e.g., the extra consumption of oxygen after exercise implies a previous oxygen debt.

Oxytocin: A hormone of the posterior pituitary which excites smooth muscle function, particularly in the uterus.

Papillae (*sing.* papilla). Structures of various shapes (round, elongated) on the tongue which contain taste-buds.

Paralysis agitans: Motor disorder characterized by extreme tonus and tremor. Also called Huntington's chorea.

Paralysis, flaccid: Paralysis characterized by flabbiness of the muscles.

Paralysis, spastic: Paralysis characterized by extreme tonus of the muscles.

Paraplegia: Paralysis of the lower half of the body.

Paraplegic: Adjective of paraplegia.

Parasympathetic nervous system (PNS): That portion of the ANS whose fibers arise from the brain stem and sacral cord (synonymous with craniosacral system).

Parasympathetic overcompensation: Hyperactivity of the PNS after a period of SNS hyperactivity.

Parathormone: The hormone of the parathyroid glands.

Parathyroid glands: A group of endocrine glands imbedded in the thyroid gland; typically four in number in man, they secrete parathormone.

Perceiving: (*See* Perception.)

Perception: The interpretation of sensation.

Perimetry: The measurement of spatial differences in the visual field; usually refers to the investigation of the limits of the field of vision for the various colors.

Peripheral nerves: That portion of the nervous system lying outside the CNS and involving three classes of neurons: (1) afferent, (2) efferent to striated muscles, and (3) efferent to smooth muscles and glands.

Peristalsis: A progressive wave of contraction in the gastrointestinal tract.

Petit mal: One of the less severe forms of epilepsy; the patient may lose consciousness very briefly, but does not fall or injure himself.

Phase: In a circular or harmonic motion, the point in the complete cycle existing at the movement of measurement, in degrees.

Photopic vision: Cone vision; vision in high illumination.

Phylogenetic scale: The ordering of

all animal forms according to evolutionary history.

Physiological psychology: The study of relationships between bodily processes and behavior.

Physiological zero: That skin temperature which feels indifferent — neither warm nor cold.

Physiology: The study of organ and tissue functions.

Pia mater: Inner meningeal layer closely adherent to the central nervous system.

Pilomotor response: Erection of surface hairs by smooth muscle contraction; gooseflesh.

Pinna: The externally visible structure of the ear; in common language, the ear.

Pitch: Psychological dimension of sound most closely related to the frequency of the sound wave stimulus; usually described as high, low, etc.

Pituitary, anterior: The adenohypophysis; the anterior portion of a small endocrine gland lying just below the brain; secretes six known hormones.

Pituitary, posterior: Neurohypophysis; the posterior portion of a small endocrine gland lying just below the brain; innervated by the hypothalamus.

Placebo: A sham drug having no physiological effect, used in research to avoid the factor of suggestion.

Placing response: (1) Visual. Placing of forelimbs on a table surface when seeing animal is brought toward the table. (2) Tactual. Placing of forelimbs as above, but with animal blindfolded and back of feet brought against the table edge.

PNS: (*See* Parasympathetic nervous system.)

Pons: That portion of the brain stem lying between the medulla and the midbrain (with the cerebellum, sometimes referred to as the metencephalon).

Positive after-potential: The wave of positive electrical potential which follows the negative potential change when a single neuron is activated.

Postcentral gyrus: The region of the parietal lobes of the cerebral cortex lying posterior to the central sulcus.

Postganglionic (neurons): Nerve fibers of the ANS which originate in outlying ganglia and terminate on effector organs.

Precentral gyrus: Gyrus of the most posterior portion of the frontal lobe; just forward of the central sulcus.

Prefrontal topectomy: Removal of small bits of prefrontal cortex.

Preganglionic (neurons): Nerve fibers of the ANS which come from the CNS and typically terminate in an outlying ganglion.

Premotor area: The area of the cerebral cortex that lies just anterior to the motor area in the frontal lobes.

Progesterone: The hormone liberated by the corpus luteum of the ovary.

Projection areas: Areas of the cortex to or from which nerve fibers lead to lower centers; usually refers to primary sensory and motor areas.

Projection fibers: Bundles of nerve fibers leading to and from specific areas of the cerebral cortex.

Prolactin: (*See* Lactogenic hormone.)

Proprioception: Senses having receptors in muscles, tendons, joints, and the nonauditory inner ear.

Prosthesis: Substitution of an artificial device for some bodily part, e.g., an artificial leg or false teeth.

Protanopia: A form of red-green color blindness in which the visibility curve is shortened at the long-wave end, and reds and greens are confused.

Protoplasm: The material of which living cells are composed.

Protozoa: Single-celled organisms.

Psychogenic: A term applied to behavior that is cerebrally initiated.

Psychology: The science of behavior of organisms.

Psychopathology: The study of severe disorders of behavior.

Psychophysics: The study of the relation between stimulus variables and sensory variables.

Psychosis: A profound disturbance of behavior, which renders an individual unable to adjust to normal daily life in an even passably satisfactory manner; true insanity.

Psychosomatic disorder: A physical disorder believed to be of psychogenic origin.

Psychosomatic medicine: That branch of medicine dealing with psychogenic influences on bodily disorders.

Psychosomatics: Connotes all psychogenic changes in bodily functions other than direct influence of one mental activity on another mental activity.

Psychotherapy: Any systematic treatment of psychological disorders which depends on interaction with a therapist; essentially a process of learning and reorientation with the help of another person.

Pupil: The opening through which light is admitted to the interior of the eye.

Pure tone: A sound wave which is a pure sine wave.

Purkinje phenomenon: The darkening of the long-wave end of the visual spectrum (reds) and the lightening of the short-wave end (blues) which accompanies the shift from cone vision to rod vision, as illumination is decreased.

Pyramidal: Adjective referring to the pyramids, or to the fiber tracts found therein. Often used to refer to all descending nerve tracts involved in voluntary movement.

Pyramidal tract: The nerve fibers running through the pyramids. Corticospinal tract.

Pyramids: Eminences on the ventral surface of the brain stem where the pyramidal tracts cross from one side to the other.

Reaction: In the behavioral sciences, action to known or inferred stimulation.

Receptor: A cell differentiated from others in terms of its increased irritability to certain stimuli.

Receptor-connector cells: Cells have both receptor and connector functions, as in the sea anemone.

Reciprocal innervation: The balance of impulses leading to the relaxation of one of a pair of antagonistic muscles as the other contracts.

Recruitment: The bringing of more and more neighboring neurons into an activity.

Recruitment, auditory: Auditory phenomenon encountered in some forms of deafness in which the RL is raised, but once a sound has exceeded the threshold it sounds very loud.

Reflex: A relatively simple, innate response to a particular stimulus.

Reflex arc: Simplest neural link from receptor to effector involving the CNS. Consists, in order, of receptor, afferent neuron, sometimes a connection neuron or neurons, efferent neuron, and effector.

Reflex, crossed extensor: Extension of the limb opposite to one showing a flexion reflex.

Reflex, extensor thrust: (*See* Extensor thrust.)

Reflex, patellar: Extension of the lower leg in response to a tap on the tendon just below the kneecap, the knee jerk.

Reflex, pupillary: Alteration in pupil size in response to changes in illumination or other stimulation.

Reflex, righting: Any reflex tending to orient an animal in relation to gravity.

Reflex, scratch: In the dog, scratching movements by a hind limb in response to stimulation of the skin of the shoulder region.

Reflex, vital: A reflex, such as breathing, directly related to the survival of the organism.

Relative refractory period: The period during which a neuron may be excited only by supraliminal stimulation; follows the absolute refractory period.

Releaser: A stimulus, usually external to an organism, which elicits more or less specific behavior with appropriate motivation.

Reproductivity (in biology): The ability of an organism, alone or in conjunction with another, to give rise to offspring.

Reticular formation: Diffuse column of gray matter running from the medulla to the diencephalon.

Retina: The innermost coat of the eye, a layer containing the visual receptors, and the primary, secondary, and tertiary neurons of the visual system.

Reverberating circuit: A closed CNS circuit in which activity may persist for some time without additional stimulation.

Rhinitis: Inflammation of the nasal membranes.

Rhodopsin: The photosensitive pigment in the rods of the retina.

RL: From the German Reiz Limen (*see* Threshold, absolute).

Rod: One of two types of visual receptors; rods function in low illumination, and are capable of producing achromatic sensations only.

Rostral: Pertaining to the head region of an organism.

Round window: Small membrane-covered opening between the inner and middle ear which permits the oval window to move in and out by relieving the pressure on the fluid of the inner ear.

Saccule: Part of the nonauditory inner ear. (*See also* Utricle and Semicircular canals.)

Sarcolemma: The membranous sheath of a striated muscle cell.

Satiation: Boredom, q.v.

Saturation: One dimension of visual sensation (or color), the fullness of a color, or lack of grayness; the depth or purity of hue.

Schizophrenia: A psychosis characterized primarily by withdrawal from reality.

Sclerotic coat: The tough outer coat of the eyeball. It becomes the cornea at the front of the eyeball.

Scotopic vision: Rod vision, vision in low illumination.

Scratch reflex: Scratching elicited, in the spinal dog, by stimulation of the skin of the chest region.

Secreting glands: Organs specialized for the liberation of chemical fluids. Duct glands secrete to the surface of the body or its cavities; the products of endocrine glands are carried in the blood stream.

Secretion: The elaboration and discharge of particular chemical substances or fluids by specialized structures in a single cell, or by specialized cells in a multicelled organism.

Sedative: A drug which depresses CNS activity in general; an agent which reduces activity of an organism.

Self: The organized set of memories, sensations, perceptions, that identify a person to himself.

Semicircular canals: Part of the nonauditory inner ear, largely concerned with acceleration. (*See also* Saccule, Utricle.)

Sensation: The uninterpreted experience accompanying afferent activity which reaches the cortical level.

Sensitizer: Essentially a motive; an internal factor which predisposes an organism to respond selectiveiy to external stimuli.

Servomechanism: A mechanism whose output is partly controlled by feeding back part of the output to the control circuits.

Sleep: State of rest and recovery, characterized in higher animals by parasympathetic dominance, reduced responsiveness, and changes in the EEG.

Smooth muscle: A muscle consisting of spindle-shaped, unstriped cells, found in the walls of viscera and blood-vessels.

Snellen chart: A chart of letters and numbers of various sizes, arranged from very large to very small, used in testing visual acuity.

SNS: (*See* Sympathetic nervous system.)

Somatogenic: A term applied to behavior that is initiated elsewhere in the body than the cerebrum.

Somatopsychic: A term sometimes employed to emphasize somatogenic effects on mental activity.

Somatopsychics: Connotes all somatogenic changes in mental activity.

Somatotrophic hormone: The growth hormone of the anterior pituitary; STH.

Somesthesis: Body sensibilities; deep and cutaneous pressure, pain, warmth, and cold.

Spasticity: Abnormally increased muscular tone as the result of disorder in the CNS.

Spermatozoan (*pl.* spermatozoa): A male germ cell.

Sphincter: A muscular ring which relaxes and contracts and thus opens or closes an orifice of the body.

Spike potential: The very brief wave of negative potential recorded as a nerve impulse passes a given point.

Spinal animal: Experimental animal in which the spinal cord has been separated from the brain.

Spinal cord: That portion of the CNS that lies within the spinal column.

Spinal reflex: Reflex obtainable when only the spinal cord is intact.

Stapes: The auditory ossicle adherent to the oval window of the inner ear. The stirrup.

Stereognosis: Tactual form perception.

STH: (*See* Somatotrophic hormone.)

Stimulus: Any energy which excites or irritates a cell.

Stress, physiological: The total physiological response that occurs in abnormally intense or prolonged stimulation.

Striated muscle: A muscle consisting of long striped fibers, usually attached to the skeleton.

Sulcus (*pl.* sulci): Term applied to the grooves on the surface of the cerebral cortex.

Summation, spatial: Increased effectiveness of a stimulus or volley of impulses as the result of a simultaneous stimulus or volley which has overlapping effects.

Summation, temporal: Increased effectiveness of a stimulus or volley of impulses as the result of an immediately preceding stimulus or volley.

Suppression, cortical: Reduction of activity in one cortical area as the result of stimulation of another.

Sympathetic ganglionic chain: A chain of interconnected neurons and ganglia of the SNS; the point of synapse of most preganglionic and postganglionic SNS neurons; lies bilaterally along the central portion of the spinal cord.

Sympathetic nervous system (SNS): That portion of the ANS whose fibers arise from the thoracic and lumbar spinal cord (synonymous with thoracico-lumbar system).

Sympatheticotonia: A term sometimes employed to designate extreme hyperfunction of the SNS.

Sympathin: The chemical substance liberated by most postganglionic axon terminals of the sympathetic nervous system (believed to exist in two forms, I and E).

Sympathin E: A chemical substance liberated by postganglionic fibers of the SNS which excites smooth muscles and glands.

Sympathin I: A chemical substance liberated by postganglionic fibers of the SNS which inhibits smooth muscles and glands.

Synapse: The point of functional contact between two neurons.

Synaptic delay: Time required for an impulse to be effective across a synapse.

Syndrome: A pattern of symptoms characterizing some disorder.

Taste-buds: Structures in the surface layers of the tongue, etc., which contain the receptors for taste.

Tectum: The colliculi usually seen as an adjective, tectal, or in combining form, tecto–, as in tectospinal tract.

Tendon: Connective tissues which serve to attach a muscle to a bone or another muscle.

Testis: The male gonad; develops male germ cells and specific hormones (*see* testosterone).

Testosterone: The major androgen secreted by the testes.

Tetanus: Sustained muscular contraction; tetanic contraction.

Thalamus: A large group of nuclei in the diencephalon.

Therblig: Term introduced by Gilbreth to designate a unit of movement in work.

Thoracico-lumbar system: The anatomical term for that portion of the ANS which involves nerve fibers coming from the thoracic and lumbar portions of the spinal cord (*see* Sympathetic nervous system).

Threshold: Usually, that value of a stimulus which can be differentiated from some standard 50 percent of the time.

Threshold, absolute: Usually, the threshold lying at the lower end of the intensity scale (synonymous with RL).

Threshold, difference: The just noticeable difference between two stimuli (synonymous with DL).

Threshold, terminal: Usually, the threshold lying at the upper end of the intensity scale (synonymous with TL).

Thyroid: An endocrine gland in the neck which secretes thyroxin.

Thyrotrophin: The hormone of the anterior pituitary which stimulates thyroid function; also called TTH.

Thyroxin: The term applied to the total hormonal output of the thyroid gland.

Timbre: Quality of a sound.

Tinnitus: Persistent noises not resulting from external stimulation; "ringing ears."

Tissue: A semistructured portion of a multicelled organism composed for the most part of cells whose functions are similar.

TL: From the German, Terminal Limen (*see* Threshold, terminal).

Tonal gaps: Frequency regions of reduced auditory sensitivity surrounded by regions of more nearly normal sensitivity.

Tonal islands: Frequency regions of auditory sensitivity surrounded by regions of impaired sensitivity.

Tonus: More or less continuous slight tension in smooth or striated muscles; sometimes known as tone.

Topographical organization: Organization in terms of place, maintenance of spatial relationships, representation of a spatial pattern.

Trichromatism: Normal color vision; three primary colors are required to match all colors.

Trophic hormone: A hormone which activates another endocrine gland.

Tropism: Inborn tendency for bodily orientation in relation to an external stimulus, such as the response of the moth to light.

TTH: (*See* Thyrotrophin.)

Tumor: An abnormal semiconfined tissue growth in an organism, which may be harmless (benign) or harmful (malignant).

Tympanum: The eardrum.

Unconditioned response: Strictly, a response which occurs to appropriate stimulation without prior conditioning.

Unconditioned stimulus: A stimulus which effects behavior without prior learning.

Unipolar cell: A neuron whose cell body has only one marked extension, the axon.

UCR: (*See* Unconditioned response.)

UCS: (*See* Unconditioned stimulus.)

Utricle: Part of the nonauditory inner ear. (*See also* Saccule and Semicircular canals.)

Vagotomy: An operation in which branches of the vagus nerves are cut.

Vagotonia: A term sometimes employed to designate extreme hyperfunction of the vagi or total PNS.

Vagus (*pl.* vagi): Cranial nerve X; an important nerve in the PNS.

Vapor pressure: A measure of the ease with which a substance volatilizes.

Vasopressin: A hormone of the posterior pituitary which increases blood pressure.

Vasopressor hormone: (*See* Vasopressin.)

Ventral: Pertaining to the belly side of an organism.

Ventral horn: The extensions of gray matter into the ventral part of the spinal cord.

Ventral root: A bundle of efferent nerve fibers leaving the spinal cord; joins with a dorsal root (q.v.) to form a spinal nerve.

Ventricle: A cavity in the interior of the brain filled with cerebrospinal fluid.

Vestibular sense: Sensation from the nonauditory inner ear.

Viscera (*pl.*) (*sing.* viscus): Internal organs and tissues.

Visual acuity: May be variously defined precisely, but, in general, refers to the ability to perceive visual spatial patterns.

Visual angle: The angle at the eye which is made if lines are projected from the edges of an object to the eye.

Vitreous humor: A viscous fluid which fills the cavity between the lens of the eye and the retina.

Voluntary movement: A movement not directly related to an antecedent external stimulus and which can ordinarily be announced beforehand by the mover.

Wave, sine: Curve obtained by plotting the angles from 0° to 360° against their sines; appears as a perfectly symmetrical wave.

Weber's law: A law in psychophysics which states that the amount by which a stimulus must be increased in order to be just noticeably different is a constant ratio of the standard stimulus.

White matter: A term used to describe those portions of the nervous system (the CNS in particular) that are given a white appearance by the myelin sheaths of masses of nerve fibers.

Work decrement: Reduction in work output by an individual following a period of work.

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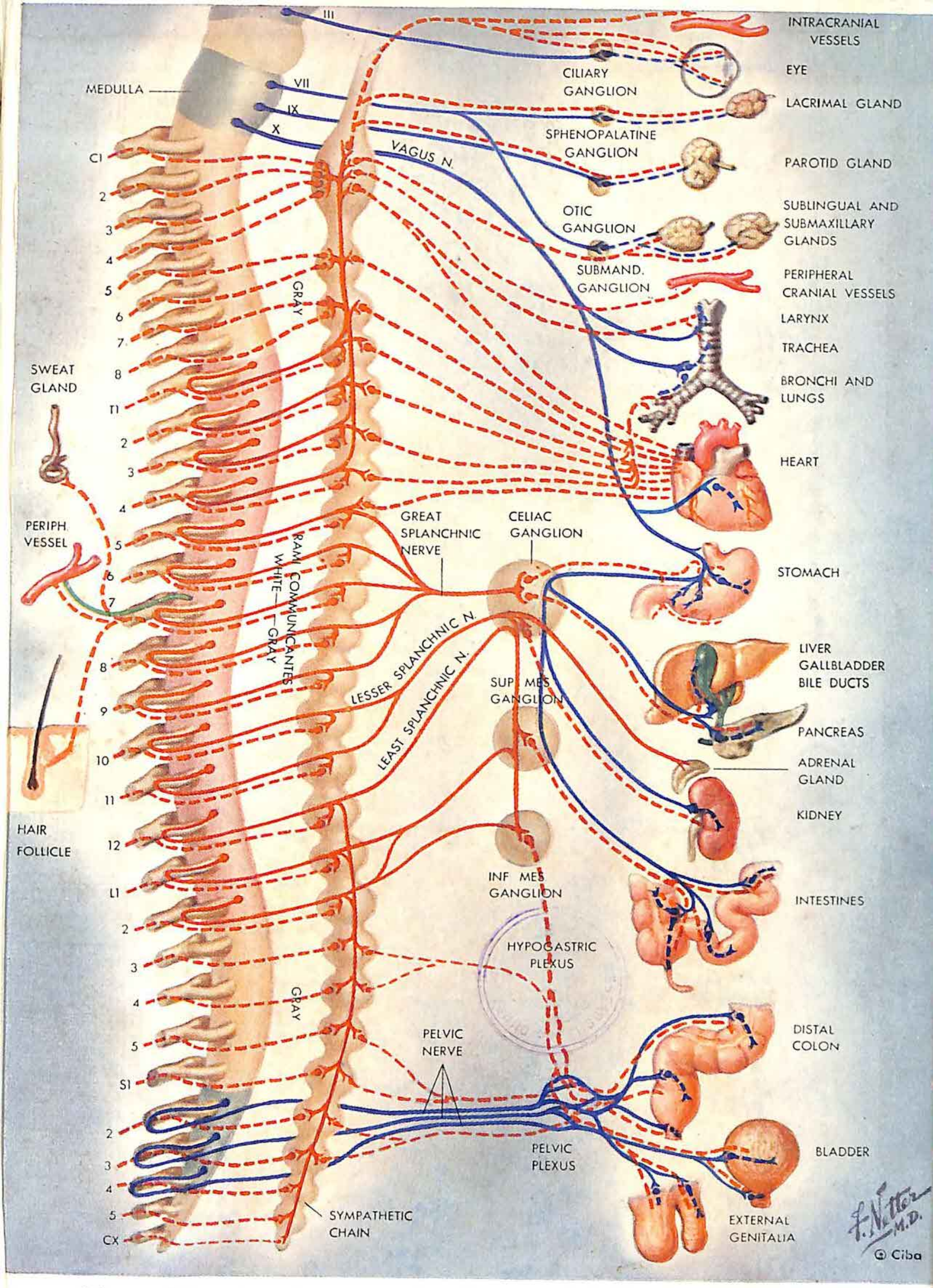

Schema of the autonomic nervous system. Parasympathetic fibers appear in blue, and sympathetic fibers in red; with preganglionic fibers shown by solid lines, and postganglionic fibers by dashed lines. (Copyright *The Ciba Collection of Medical Illustrations.*)